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## Original Communications

### SPECIAL CONSIDERATIONS RELATING TO SURGICAL CLOSURE OF LARGE UPPER LOBE TUBERCULOUS CAVITIES*

JOHN ALEXANDER, M.D., ANN ARBOR, MICHIGAN

NONCLOSURE of large or stiff-walled cavities in the upper third of the lung is the most frequent cause of failure to effect cure of tuberculosis by artificial pneumothorax or surgical operation. Increasing experience with the surgery of pulmonary tuberculosis has demonstrated relatively safe means whereby the frequency of such failures may be greatly reduced.

Moderate relaxation of a lung containing predominantly fibrotic lesions by phrenicectomy, partial pneumothorax, a small extrapleural pneumolysis, or a small paravertebral thoracoplasty, or by a combination of these, can reasonably be relied upon to close soft-walled cavities of not great size by contraction of the elastic and fibrous tissue around them; occasionally this occurs spontaneously. But large or stiff-walled cavities often require more than moderate relaxation. They may require actual compression or at least relaxation of the lung around a very great part of their circumference.

A total pneumothorax without pleural adhesions and in the presence of a nonmobile mediastinum against which the lung may be pressed by positive intrapleural air pressure, is excellent treatment for such cavities as are under consideration. But it is unusual to obtain a total pneumothorax over such extensive lesions. In only a minority of cases can a partial and inadequate pneumothorax be made complete by cauterization of adhesions under thoracoscopic control or by open thoracotomy, or be made wholly effective by a supplementary phrenicectomy, with or without scaleniectomy. Extrapleural paravertebral thoracoplasty, when not

*From the Department of Surgery, University of Michigan Medical School and the University Hospital.

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contraindicated, must next be undertaken if the preceding measures have failed to close the cavity after a wait of several months to give contraction of fibrous tissue its chance.

A thoracoplasty in which only short lengths of ribs are resected should not be used for large or thick-walled cavities. Such an operation frequently fails to close the cavity. Brunner[1] reported in 1924 that 14 per cent of the last 117 thoracoplasty patients at the Sauerbruch Clinic required reoperation, usually further resections paravertebrally in the field of the regenerated ribs, which is technically more dangerous than was the original operation. Sauerbruch[2] tends to resect shorter segments of ribs than do many American surgeons, so as to make safer the one-stage operation that he prefers, although he[3] approves the principle of varying the lengths of ribs to be resected to correspond to the requirements of each patient. Brauer,[4] on the other hand, usually resects in one stage, great lengths of ribs for small as well as large cavities, but the majority of surgeons are opposed to this because it is unnecessary for small cavities and far more dangerous than a small or a many-staged large thoracoplasty.

Very great lengths of the ribs overlying large or stiff-walled cavities should be resected at the primary operation, the lengths varying somewhat according to the rigidity of the pleurae and the mobility of the mediastinum. Such an operation performed in one or even two stages would be highly dangerous for the average patient whose cardiocirculatory system and general resistance had been damaged by prolonged severe tuberculosis. Performed in three or more stages, preferably at intervals of two weeks, the operation becomes relatively safe. Hedblom[5] has made a notable contribution to the surgical treatment of pulmonary tuberculosis in demonstrating the advantages of many-staged operations. Many American and other surgeons, following his teaching, are now routinely using two, three, four or more stages in cases for which they would formerly have used one or two.

There appears to be universal difficulty in exposing the axillary portion of the ribs through a paravertebral incision that does not extend considerably beyond the inferior angle of the scapula. Most thoracic surgeons do not attempt to resect more than from 2 to 5 cm. of the first rib, although recently Ziegler[6] and Proust and Maurer[7] have removed much greater lengths, as has Sauerbruch[3] with rongeurs. Lilienthal[8] and many others have written of the relatively inaccessible position of the deep-lying first rib and of the difficulty of resecting, through a proper paravertebral incision that spares the anterior 5 cm. of the trapezius, even a moderate length of the first rib safely because the brachial plexus and subclavian artery lie directly upon it. In recent years there have appeared a number of articles[9] describing a variety of special incisions above the clavicle, above the scapula, or in the axilla, designed to make partial resection of the first, or first and second ribs simpler and safer.

For greater ease in resecting the posterolateral segments of the subscapular ribs, Brauer[10] uses an axillary incision in conjunction with a paravertebral incision. Through two incisions the ribs are freed from their periosteum and divided laterally and paravertebrally and then with special periosteal elevators, operated on both from before and behind, those portions of the ribs underlying the scapula are blindly freed from their periosteum and pulled out. The blind phase of this operation exposes the pleura and intercostal vessels to considerable danger of being torn.

Several years ago I devised a very simple maneuver that gives surprisingly easy access to the first rib and to the subscapular ribs. A thoracic surgeon of great experience with thoracoplasty was a visitor in my operating room not long ago and was unable to believe that a rib that was broadly exposed without strong retraction of the soft parts could be the first, which it was. I have steadily used the method with entire satisfaction for all thoracoplasties that have required resection of considerable lengths of the upper ribs.

The usual J-shaped paravertebral incision is used, beginning about 5 cm. below and behind the anterior border of the trapezius and ending close to the inferior angle of the scapula. After the trapezius and rhomboid muscles have been divided, an assistant lifts the vertebral border of the scapula away from the thoracic wall and the surgeon divides the areolar tissue that covers the anterior surface of the serratus magnus muscle and then with scalpel or scissors cuts through this muscle close to its origin from the second rib where the muscle presents a convenient posterior angulation. The surgeon next introduces his finger through the small incision and pushes away the axillary fat and axillary vein from the posterolateral surface of the muscle and then continues the incision in the muscle upward to the first intercostal space, while holding away the axillary fat and vein with his finger, and downward to the fifth or sixth rib. Usually only one or two small vessels are divided and are easily secured and ligated; rarely, a large artery is divided near the second rib. This whole procedure occupies only a few moments. The scapula may now be easily lifted well posteriorly and laterally away from the ribs whose resection is thereby greatly facilitated (Fig. 1). In none of my patients have I seen any evidence that division of the serratus magnus in any way hindered return of excellent function of the shoulder girdle. The divided edge of the muscle probably rapidly adheres to the decostalized thoracic wall.

In a number of patients with very large upper lobe cavities I have resected approximately 10 cm. of the first rib, beginning somewhat mesial to the tip of the transverse process of the vertebra which was also resected for maximal effect upon that portion of the cavity lying in the costovertebral gutter. Removal of a greater length of the first rib would require division of the rib anterior to the tendon of the scalenus anticus

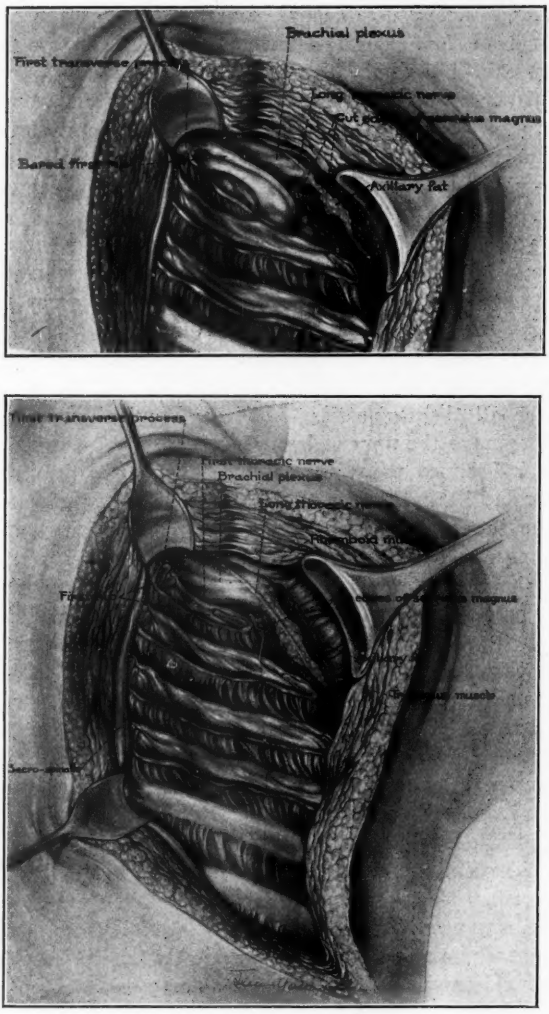


Fig. 1.—Division of the upper five origins of the serratus magnus has given excellent exposure of the axillary portions of the upper ribs, including the first, through the customary paravertebral incision in which the anterior 4 to 6 cm. of the trapezius have not been divided. The axillary fat, the brachial plexus and (sometimes) the long thoracic nerve are plainly visible. In the upper drawing the posterolateral portions of the second and third ribs have been extensively resected and 7 to 10 cm. of the first, having been freed from its periosteum, is about to be removed. If there is doubt about the patient's condition, only three ribs are resected (and occasionally only two). Rarely are more than four ribs removed as extensively as illustrated in the lower drawing, at the first stage of the thoracoplasty.

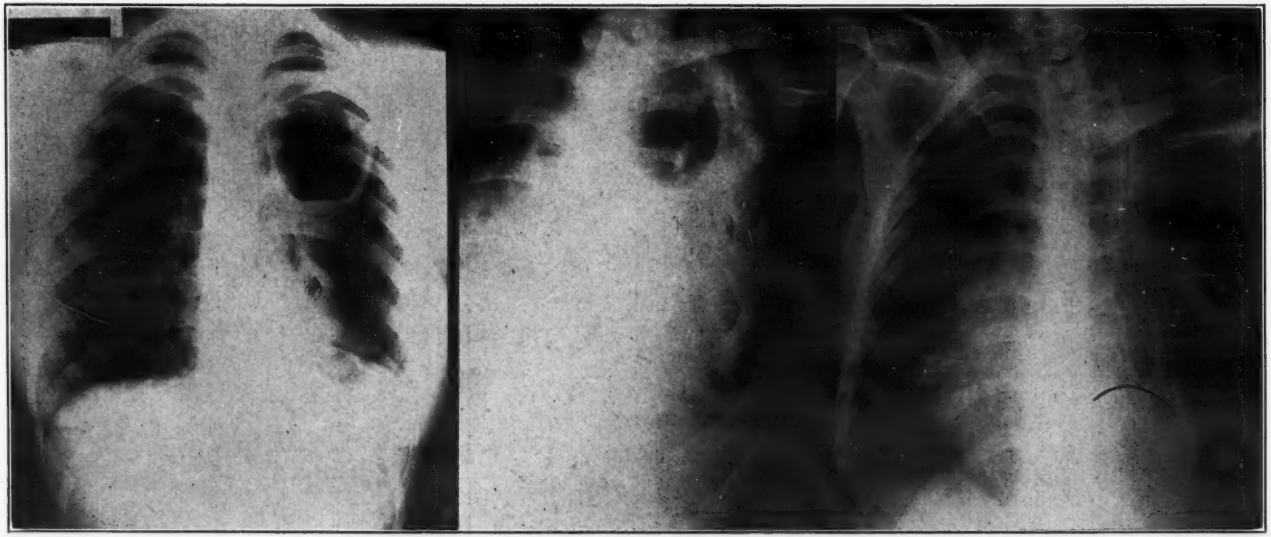

Fig. 2.—*Left.* Bilateral tuberculosis with mammoth (11 by 7.8 cm.) cavity 15 months after onset of disease. Ineffective artificial pneumothorax. Twenty-one-year-old woman. One hundred and eighty cubic centimeters positive sputum daily. Great loss of weight. Tuberculous laryngitis. Symptoms of tuberculous enteritis.

*Center.* Six months after phrenicectomy (4.7 cm. rise of diaphragm, but cavity larger) and 2 weeks after completion of very extensive (175 cm.) posterolateral eleven-rib thoracoplasty in four stages. Cavity greatly reduced in size but still large. It is being kept from closing by the anterolateral stumps of the upper 3 ribs (arrows) that remain in spite of posterolateral removal of 10.5 cm. of 1st rib, 14.5 of 2nd and 17.5 of 3rd.

*Right.* Apparent complete closure of cavity 3 months after complementary two-stage anterolateral thoracoplasty of the remaining 25 cm. of the upper 3 ribs and cartilages and the left half of the sternum from the sternoclavicular joint to the 4th cartilage which was divided close to the sternum to allow 4th anterolateral stump to fall inward. See Figs. 8 and 12. Within 1 year of operation patient had gained 40 pounds, lost symptoms of laryngitis and enteritis, and sputum was reduced to 5 c.c. or less daily and negative. Two years after operation, this condition was maintained; sputum 0 to 5 c.c. (except during a "cold") and always negative.

which might be unwise because of the possibility of the sharp edges of the cut end of the rib tearing the axillary vein. In one of my patients, a woman of average girth, with a mammoth pulmonary cavity from whom 10.5 cm. of the first rib were removed, the following centimeter lengths of the next ten ribs were resected in four stages: 14.5, 17.5, 18, 19, 21, 20, 18, 17, 13, 10, a total of 178.5 cm. (Fig. 2). In another patient a total of 216 cm. was resected posterolaterally. The average total posterolateral resections in 13 patients (one of 14 patients who had both posterolateral and anterolateral thoracoplasties for pulmonary tuberculosis, needed to have only seven ribs resected posterolaterally and is, therefore, not in-

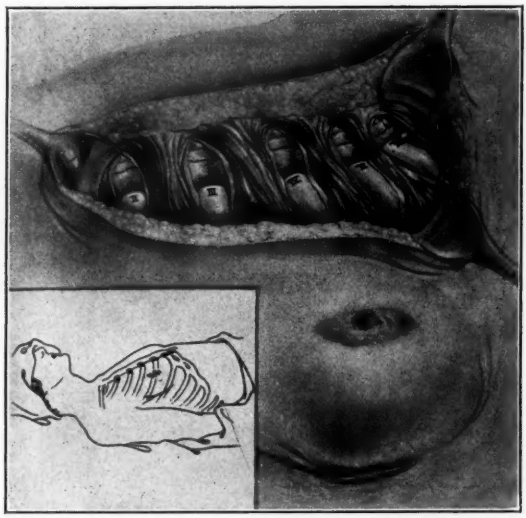

Fig. 3.—Parasternal chondrectomy (II-VII). This operation allows the anterolateral rib stumps remaining after a subsequently performed posterolateral thoracoplasty to fall inward far more than if this movement depended on the bending of intact cartilages. It is used for maximal compression of large or stiff-walled pulmonary cavities, short of the addition of a deforming complete anterolateral thoracoplasty. It is useful especially for patients old enough to have presumably relatively stiff costal cartilages. On the left side, the 2 cm. chondral resections should be made at the left border of the heart so as not to press upon it, instead of close to the sternum. See Figs. 4 and 5.

cluded in this group) who had sufficiently large cavities to require a supplementary anterolateral thoracoplasty were 148 cm. The average length of the first rib resected posterolaterally in these patients was 8.4 cm. and of the second rib, 12.6 cm. The average total length of the first rib and cartilage (from the tip of the transverse process) as measured in three skeletons was 11.23 cm. and of the second rib and cartilage, 20.25 cm. These resections usually included the ends of the transverse processes of the vertebrae and the underlying portions of the ribs so as to increase collapse of the lung in the costovertebral gutter. Brauer[11] in his "subscapular thoracoplasty," resects approximately the following

lengths: ribs I-X, 3.5 cm., 8.8, 12.8, 13.5, 15, 15, 15, 15, 14, 11.5, a total of 124.1 cm.

It would not be difficult to remove still greater lengths of the first and other ribs because their anterior ends and cartilages lie in the sagittal plane of the paravertebral wound and are therefore approximately as accessible as the most lateral portions of the same ribs. If greater lengths than those specifically cited need to be removed I believe that it is safer to postpone this phase of the operation to another stage and then operate by the anterolateral thoracoplasty technic that will presently be described.

Pressure upon the anterolateral costal stumps after paravertebral thoracoplasty, especially during early convalescence while the tissues in the field of operation are soft and pliable, often results in a surprising posterior and mesial swing of the stumps, pivoting at their elastic carti-

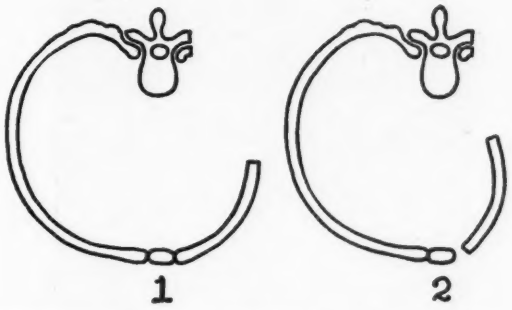


Fig. 4.—Diagrammatic explanation of operation described under Fig. 3. For illustration of *1* see Fig. 2, center, and of *2*, Fig. 5.

lages. Such pressure probably often results in closure of a cavity that otherwise could scarcely have been expected to close.

Joseph Gale[12] has recently suggested a revival and modification of the operations of Eden and Robinson[13] and Wilms.[14] Short (2 cm.) parasternal pieces of the cartilages should be resected (Figs. 3 and 4) at an operation under local anesthesia in advance of a paravertebral thoracoplasty so as to cause the anterolateral ribs and cartilages remaining after the paravertebral operation to fall in and diminish pulmonary volume to the greatest possible degree, short of addition of a true anterolateral thoracoplasty. This preliminary parasternal chondrectomy is intended for large cavities especially in patients who are middle-aged or older and in whom the cartilages are therefore presumably less elastic than in younger persons and less likely to bend to allow the anterolateral rib stumps to fall in. I have so far performed this operation only once; it resulted in a pulmonary collapse (Fig. 5) that was decidedly in excess of what would have been expected in the 34-year-old patient, had only a

paravertebral thoracoplasty been performed. Even for young patients with large cavities I expect to use this operation frequently and I believe that it will greatly improve the results of posterior thoracoplasty and probably often spare the patient an anterolateral thoracoplasty. On the left side it is probably better to make the short resections of the third to seventh cartilages or ribs at the left border of the heart, instead of parasternally, so that the heart will not be pressed upon as a fulcrum. The second cartilage should be resected parasternally.

Pressure upon the anterolateral costal stumps after a paravertebral thoracoplasty is applied in three ways: (1) Immediately after operation

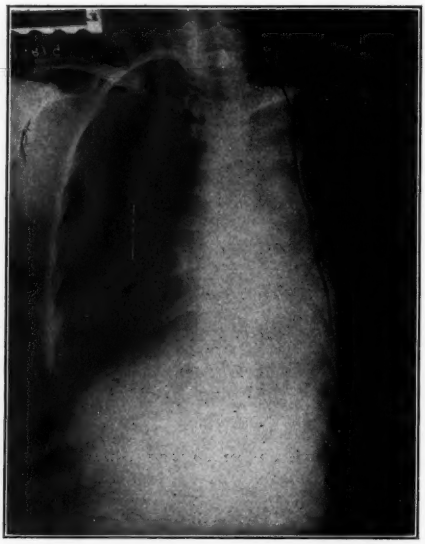

Fig. 5.—Six weeks after completion of phrenicectomy, parasternal chondrectomy (Fig. 3) and 3-stage posterolateral thoracoplasty. The preliminary chondrectomy has apparently resulted in a far greater compression of the lung in this 34-year-old woman with presumably relatively rigid costal cartilages, than if posterolateral thoracoplasty alone had been performed. Seven-year tuberculosis history. Before operation, multiple cavities had nearly destroyed the upper lobe; 34 to 66 gm. positive sputum. Now 7 to 12 gm. negative sputum.

the patient is placed in bed lying with his operated side upon a pillow that reaches high in the axilla. This is well tolerated while the local anesthesia is still active or immediately after gas anesthesia, and once having assumed this position the patient is better able to return to it. The position should not be used immediately after operation for any patient whose cardiorespiratory function might be seriously compromised by the additional pressure. The patient may alternate between the lateral and supine positions. (2) When lying on his back he should keep, for as long a time as tolerable, a shot bag upon the ribs beneath the clavicle. To begin with, the bag should weigh three pounds and heavier

ones be gradually substituted. (3) Some days after paravertebral resection of the ribs below the third or fourth, a pad pulled upon by elastic straps should be worn in the axilla (Warriner Woodruff[15]).

In spite of a very extensive posterolateral thoracoplasty (even when preceded by parasternal chondrectomy) followed by pressure as described, not a few large or stiff-walled cavities remain so large several weeks after operation that there seems no reasonable chance that they will close without some supplementary operation (Fig. 2). Under such circumstances three choices are available: (1) Delaying decision as to necessity for a supplementary operation for from eight to twelve months. (2) Extrapleural pneumolysis. (3) Anterolateral thoracoplasty.

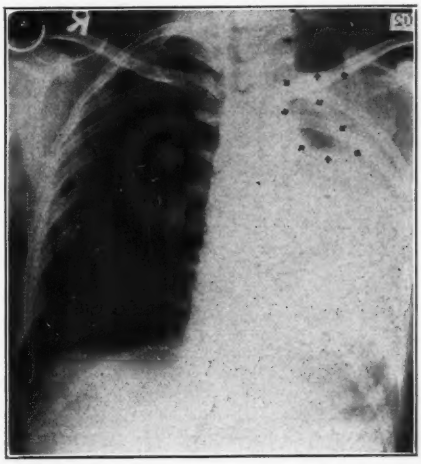

Fig. 6.—Nine days after removal of posterolateral portions of 1st, 2nd and 3rd ribs. The oval cavity, which before operation was nearly round and lay both above and below the clavicle, has been pushed entirely below the clavicle and made much smaller. The upper arrows outline a collection of air which was trapped between the rib beds and the scapula and its attached muscles when the incision was closed. Below this air is a fluid level of wound secretion. Such a collection of air and serum is very common after resection of fewer than 6 upper ribs and it may be thought to be the pulmonary cavity itself if this has become closed or is invisible. The extracostal space is formed by collapse and depression of the upper lung (a sort of extrapleural pneumolysis) and is surprisingly great when the costal resections are extensive. After the upper 6 or 7 ribs have been removed the scapula will fall in and obliterate the dead space holding the air and serum.

(1) Wait from eight to twelve months, as Sauerbruch[3] does, in the hope that progressive formation and contraction of fibrous tissue will gradually close the relaxed cavity. The chance of delayed closure of the cavity should be increased if the periosteum of the resected ribs lying over and somewhat beyond the cavity has been resected or destroyed by Zenker's solution (Jerome Head[16]) at the time of the paravertebral operation so as to prevent fixation of the thoracic wall by regeneration of ribs before the fibrosed lung has shrunken to the limit. Permanent decostalization of the posterior portion of the upper thoracic

wall should not compromise respiratory function or activate the tuberculous lesions, because the overlying scapula prevents undue mobility of the thoracic wall. It is doubtful, however, whether the periosteum should be destroyed inferior or anterior to the scapula because of the harmful effects of paradoxic thoracic wall movements. If closure of the cavity does not occur within from eight to twelve months it is Sauerbruch's practice to reoperate paravertebrally, resecting the regenerated ribs and

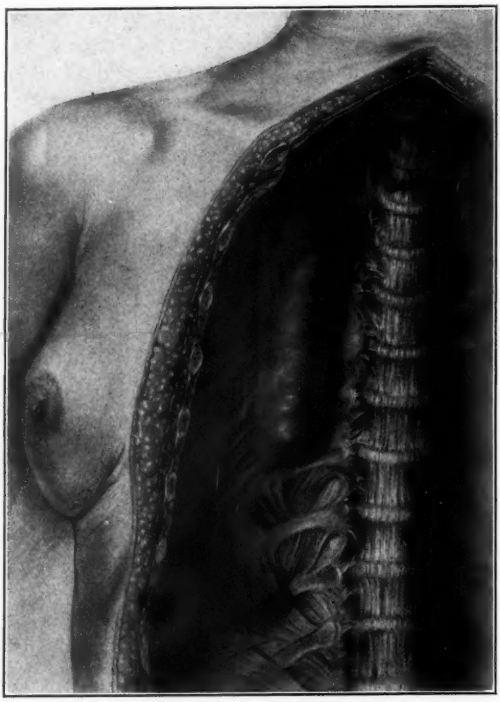

Fig. 7.—Drawing of necropsy specimen showing the large cylindrical mass of intercostal structures that bulges forward between the divided ends of the ribs after a medium-sized extrapleural thoracoplasty (86 cm. of the upper 7 ribs were resected). This mass of tissue reduces the volume of the lung in the costovertebral gutter where reduction is especially desirable.

parts of the adjacent anterior stumps and perhaps at the same time to perform an extrapleural pneumolysis with gauze tamponade, or he may carry out an anterolateral thoracoplasty.

I feel that it is proper to defer decision about performing a supplementary operation if there is a reasonably good chance that final closure will occur after paravertebral thoracoplasty alone, but if this is unlikely, even though a possibility, I believe that the ultimate prognosis will be

better if a supplementary anterolateral thoracoplasty is performed within a very few weeks after paravertebral resection of the upper ribs; see (3).

(2) Perform an extrapleural pneumolysis with paraffin or muscle or rubber bag filling. I prefer anterolateral thoracoplasty because compression of a cavity can be progressively increased with an external pressure pad and it has, therefore, a better chance of closing a resisting cavity than would a paraffin or muscle filling of safe size. A thoracoplasty, being a definitive operation, is preferable to a rubber bag pneumolysis which should be reserved for patients in whom thoracoplasty is contraindicated. I have, however, occasionally found extrapleural pneumolysis useful in displacing a large cavity from the protecting concavity of the anterolateral portion of the thoracic wall and also from the costovertebral gutter before performing a paravertebral thoracoplasty. It has been my experience, in common with that of several others who have used extrapleural pneumolysis many times as an independent operation, that a filling of safe size only rarely closes a large or stiff-walled cavity (it is of real value for strictly localized lesions with only small or medium sized cavities) for the reason that the mediastinum may move over and that there is no firm tissue inferiorly against which the filling may press the lung. The cavity, therefore, tends to shift mesially and inferiorly and to become only partly closed. On the contrary, when pneumolysis is used in conjunction with a thoracoplasty of ten or eleven ribs the collapsed thoracic wall tends to keep an open cavity from being pushed downward by the paraffin filling.

(3) Anterolateral thoracoplasty has notable advantages as an operation supplementary to an extensive paravertebral thoracoplasty. Some cavities, after the latter operation, appear in a roentgenogram to be directly kept from closure by the anterolateral portions of the ribs and their cartilages (Fig. 2). Removal of these, followed by the wearing of an external pressure pad, is capable of causing practically total obliteration of the upper hemithoracic cavity (Fig. 2). This will close any upper lobe pulmonary cavity that is capable of closure by compression, provided, of course, that the mediastinum is sufficiently fixed to offer adequate counterpressure. It is obviously true that an anterolateral thoracoplasty that removes the ribs, but not the cartilages, may cause sufficient compression of the lung to close a large or stiff-walled cavity that has resisted closure by a posterolateral thoracoplasty. It is equally true that a cavity may be so large and stiff-walled as to require for its complete closure the utmost possible compression; this cannot be got if the cartilages are not resected. Which operation had better be performed, with particular reference to the lesser deformity when the cartilages are not resected, is a difficult question. My feeling is that, in view of the ultimately poor prognosis if the cavity cannot be completely closed, the maximal compression should be given to any stiff-walled cavity that has

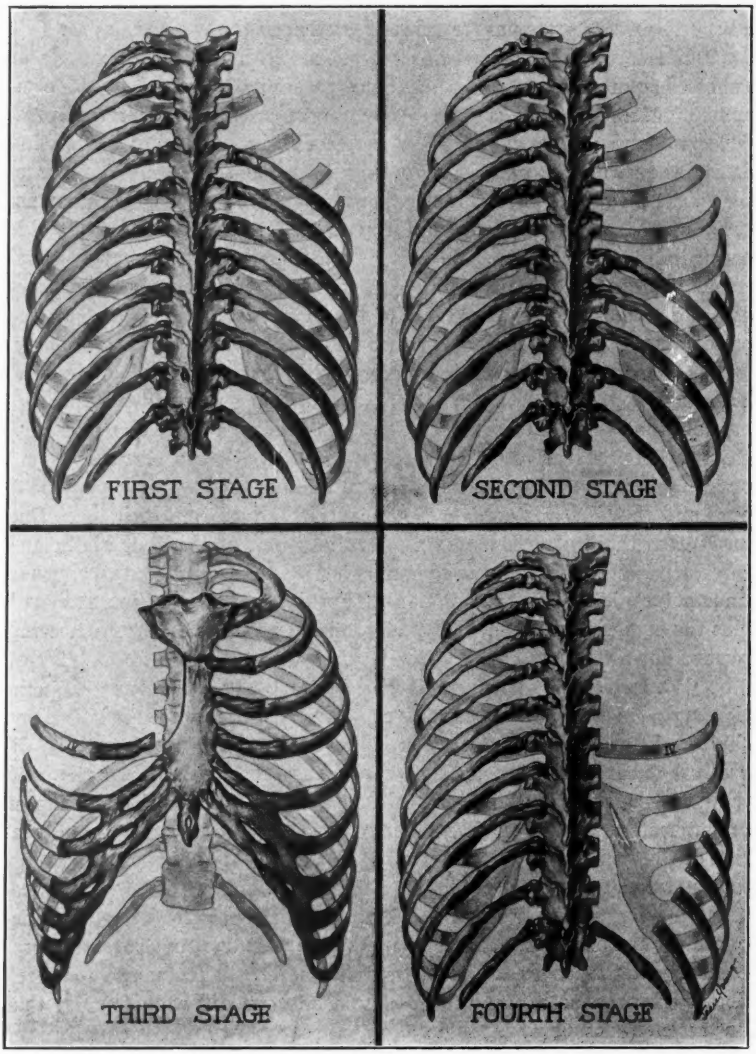


Fig. 8.—The 4 stages of a very extensive posterolateral and anterolateral thoracoplasty for a huge upper lobe cavity (Fig. 2). If the patient's condition were not good the posterior thoracoplasty might be divided into 4 or more stages and the anterior into 2. Posteriorly the ribs have been resected mesial to the tips of the transverse processes which have also been resected to give maximal compression of the lung in the costovertebral gutter. Anteriorly a portion of the right upper sternum has been removed so as to obtain maximal compression of a cavity extending to the mesial limit of the lung (Fig. 2). A portion of the fourth cartilage has been removed to aid the anterolateral portion of the 4th rib, which is well below the bottom of the cavity, in swinging posteriorly and mesially. Additional anterolateral ribs and cartilages might be removed if the depth of the cavity demanded this. The falling in of the anterolateral stumps is not represented in these drawings but is in Fig. 2.

not become a small one shortly after an extensive posterolateral thoracoplasty.

When properly performed, the operation causes less discomfort to the patient than any stage of the paravertebral operation. The two operations combined have, therefore, the great advantage of the old Brauer-Friedrich operation, without its dangers. Resection of the anterior portion of the first rib is undoubtedly dangerous, because of the close connection of the first rib and subclavian vessels but I do not recall any report of an accident by other surgeons and I have personally had none in fourteen cases.

After a paravertebral thoracoplasty the intact anterolateral portions of the ribs keep the decostalized paravertebral periosteum from dropping as far mesially as it would if the entire length of the ribs had been resected. It follows that the ribs that regenerate from that periosteum are

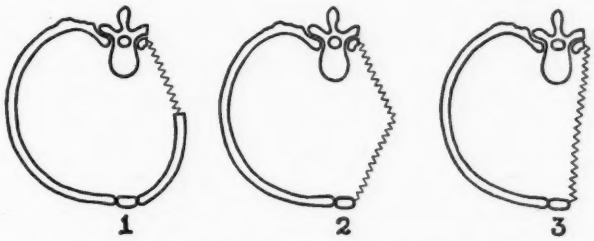


Fig. 9.—To illustrate why performance of an anterolateral thoracoplasty after regeneration of the ribs in the field of a preliminary posterolateral thoracoplasty, results in incomplete collapse of the lung (*2*). When the anterior operation is performed before the posterior periosteum stiffens, the resulting collapse is complete (*3*). *1* shows a regenerated posterior rib before the anterolateral thoracoplasty has been performed. The zigzag lines represent regenerated ribs.

also partly held out (Fig. 9). If the anterolateral thoracoplasty is not performed until after this regeneration has occurred, it is obvious that the periosteum of the anterolateral portions of the ribs will, in turn, be unable to fall as far mesially as if there had not been regeneration of the posterolateral portions of the ribs. In this event collapse of the thoracic wall will be less than maximal and the pulmonary cavity may remain open. This occurred in three of my early cases and since then I have performed the anterolateral operation, when the indications were clear to proceed without awaiting possible closure of the cavity, within from two to five weeks of paravertebral resection of the upper ribs. This point has previously been stressed by Welles.[17] As it is my practice to resect, paravertebrally, the upper ribs before the lower ones and as in these sick patients with large cavities I usually resect only the first three ribs at the first stage and the next three or four ribs at the second stage approximately two weeks later. I have recently been performing the anterolateral operation as the third stage, and resecting the remaining

paravertebral ribs at the fourth or fourth and fifth stages. This method of procedure permits tremendous collapse of the upper thoracic wall before any bone regenerates from the periosteum (Fig. 2). Painting the periosteum of the ribs resected paravertebrally with Zenker's solution may be found to be effective in preventing costal regeneration so that performance of anterolateral thoracoplasty need not be hurried.

I am reporting my technic for anterolateral thoracoplasty (Fig. 10) because it seems to me simpler and more thorough than the various other technics that I have seen described. Most surgeons who use an axillary incision resect the ribs only as far forward as the costochondral junctions, which Sauerbruch[3] states "can be reached without great difficulty," and that the first rib can be reached if the incision is carried high in the axilla. If the patient is stout and short he prefers an incision over the costochondral junctions, beginning at the middle of the clavicle; he then splits the pectoralis major over each rib and finds the first rib difficult to resect. Gravesen[18] found that the first rib was usually too inaccessible to resect. Hedblom[5] resects through a midaxillary incision the upper nine or ten ribs in two stages from their costochondral junctions to the site of their division at the time of the posterior thoracoplasty. Welles[17] also resects only from the costochondral junctions and usually removes the second to the sixth or seventh, inclusive, but not the first.

Brunner[19] who prefers a supplementary anterolateral thoracoplasty of the upper six, seven or eight ribs to extrapleural pneumolysis, employs an incision across the pectoralis major if he intends including the first rib and splits the muscle over each rib. He does not resect the first if it has been resected paravertebrally. If he does not take the first rib he makes an axillary incision, and never exceeds 9 cm. in the resection of any rib. Bérard and Dumarest[20] make a vertical incision over the costochondral junctions, making their resections in much the same way as Wilms[14] did originally in 1911. Wilms resected 2 or 3 cm. of the first rib and 4 or 5 cm. of the next four, five or six cartilages and adjoining ribs. A further discussion of the early types of parasternal thoracoplasty appears in a book by Alexander.[21]

The technic I now use is as follows: The incision (Fig. 10) is semilunar and in the case of a woman, passes lateral to the breast. An incision directly over the anterior axillary fold permits less easy mesial retraction of the pectoralis major and is apt to be followed by adduction contracture of the arm. The pectoralis major is retracted anteriorly and mesially and the origins of the pectoralis minor are divided. When the arm, which is held throughout the operation by an aide, is abducted and mesially flexed at the shoulder joint, the pectoralis major is relaxed so that it may be retracted to afford entirely satisfactory access to the upper ribs all the way to the sternum. Exposure of the cartilages may be increased by cross-cutting for 3 or 4 cm. the lateral border of this muscle at the lower end of the incision, but this is not often needed.

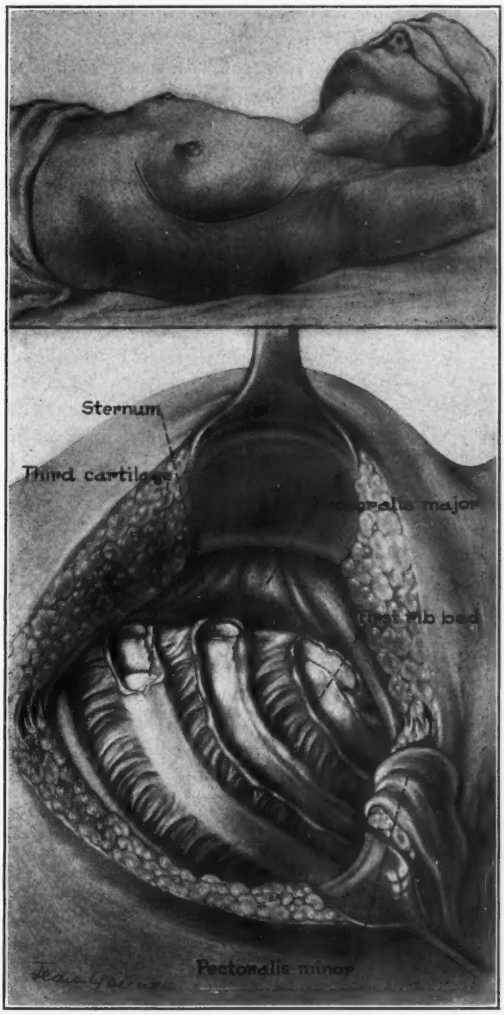


Fig. 10.—Anterolateral thoracoplasty, including costal cartilages, for a large or stiff-walled cavity that an extensive posterolateral thoracoplasty has failed to close. Abduction of the arm exposes the first rib by lifting the clavicle away from it, while mesial flexion of the arm permits easy retraction of the pectoralis major from the cartilages. The 1st and 2nd ribs have been resected from the sternum to where they had been divided during the posterior thoracoplasty, and a piece of the 3rd cartilage has been removed so as to allow the 3rd rib to swing backward and inward and yet protect the heart.

Anterolaterally I have never found it necessary (except in cases of extensive empyema) to resect more than the upper four ribs and divide the cartilage of the fifth. Resection of the upper two ribs and division of the third cartilage usually suffice to close the average large cavity, after an extensive complete paravertebral thoracoplasty has failed to do so. The cartilage of the rib that lies below the lower limit of the cavity is divided, so as to cause the remains of that rib to hinge at the place of division and to fall posteriorly and mesially and relax the lung beneath it to some degree. The dropping of the rib leaves a gradual slope from the lower intact ribs to the bottom of the depression of the sunken thoracic wall from which the upper ribs have been resected. Leaving the rib provides bony protection to the base of the heart in left-sided operations.

The first step in resection of the ribs is to free the second rib and cartilage from their periosteum and perichondrium, special care being taken not to tear the pleura at the costochondral junction. Next the cartilage is divided close up to the sternum, any remaining stump of cartilage being removed to the sternum with rongeurs, and the anterior end of the rib is held in the hand while the axillary portion is gently separated from its periosteum with a periosteal elevator. Thanks to the previous extensive paravertebral resections of the upper ribs, it is not difficult to reach, usually under direct vision, the site of division of the rib from behind. When the operation is not performed until after new rib has regenerated from the posterior periosteum, the anterolateral portion of the rib does not come away as soon as separation of the periosteum has reached the site of previous division but the cordlike new rib must be divided as far posteriorly as possible after cautiously stripping off its periosteum.

After the second rib has been removed, the first is approached. With the shoulder in the normal position very little of the first rib appears beneath the clavicle, but when the arm and shoulder and clavicle are pulled upward there is excellent access to this rib. Raising of the arm and shoulder also lifts the subclavian-axillary vein which reduces the danger of its being torn. The close proximity of this vein and the periosteum of the first rib must be kept constantly in mind while operating upon this rib. The inferior edge and then the anterior and posterior surfaces, and finally the superior edge of the rib, are freed from periosteum and from the origin of the subclavius muscle and from Sibson's fascia, and the perichondrium is then stripped from the cartilage as far forward as the costoclavicular ligament permits and then the cartilage is divided cautiously with bone shears obliquely from its inferior and mesial edge to its superior and lateral edge. The remaining posterior portion of the rib is removed as was the corresponding portion of the second rib. Next is removed all of the cartilage of the first rib that lies between the

sternum and the place where the cartilage had been divided with shears, except that part that lies behind the sternoclavicular joint. This is most conveniently done with rongeurs after careful stripping of the perichondrium posteriorly so as to protect the innominate vein.

If resection of the third rib is indicated, this is done next. Finally, 1 or 2 cm. of the cartilage, next below the rib last resected, is removed subperichondrally close to the sternum.

In three patients with very large cavities, for which the greatest possible collapse of the thoracic wall was desirable, a 2 cm. width of the

Fig. 11.—Thick sponge rubber which is pressed by elastic straps into the subclavicular defect created by an anterolateral thoracoplasty so as to compress to the greatest possible extent an unusually large or stiff-walled cavity, which an extensive posterolateral thoracoplasty has failed to close. Before performing so extensive an anterolateral thoracoplasty, the patient is asked to choose between an open pulmonary cavity and an ugly external deformity; not one has chosen the former.

sternum from the level of the sternoclavicular joint to the fifth cartilage was resected in addition to the cartilages.

A few approximation sutures are placed in the subcutaneous fascia of the wound and the skin is closed, leaving a 36-hour tube drain. A snug Elastikon dressing is placed so as to stabilize the decostalized anterior thoracic wall, but heavy pressure should not be used during the first days after operation because of the danger of causing cardiocirculatory decompensation from too sudden pressure upon and displacement of the mediastinal organs. If the patient's condition should be poor be-

fore or during the operation, the anterolateral thoracoplasty should be divided into two stages.

Pressure of a thick dressing should be gradually increased during the next ten days and then a special brace[15] should be fitted (Fig. 11). The deep pad of this brace is made of several layers of sponge rubber, cemented together, cut to the proper shape and covered with flannel. The pad is pressed against the decostalized portion of the anterior thoracic wall by means of heavy elastic straps. This brace should be worn constantly for at least three months or until new bone has regenerated from the periosteum that will fix the thoracic wall in a fully collapsed and

Fig. 12.—Ten months after operation illustrated by Fig. 2. Practically no deformity as viewed from behind owing to shoulders being held the same distance from the midline by the clavicles. The extensive anterolateral thoracoplasty, which was necessary for this patient, leaves an ugly deformity which may be concealed by a pad under the clothing.

immobile position. For some unexplained reason regeneration has failed to occur in several of my patients, although the resections were made subperiosteally and subchondrally, and the patients have been obliged to continue wearing the brace.

The purpose of the brace is, in addition to immobilization, to cause the thoracic wall to sink in as deeply as possible so as to give the cavity the very best possible chance of becoming closed. My two largest pulmonary cavities measured in their greatest diameters 12.4 by 7.8 cm. and 11 by 8 cm., respectively; both were apparently completely closed by the combined posterolateral and anterolateral thoracoplasties described in this article. The deep depression that occurs beneath the clavicle is ugly (Figs. 11 and 12). I always tell my patients before operation what

the deformity will be and say that they must choose between a depression of the thoracic wall and a cavity within the lung. Without exception they have chosen the former.

Most medium-sized and some large upper lobe cavities become completely closed after extensive posterolateral thoracoplasty and therefore the indications for a supplementary anterolateral thoracoplasty are relatively few. Among approximately 100 patients who have had posterior thoracoplasty for pulmonary tuberculosis it has been necessary to use this operation in only 14 cases, the first anterolateral thoracoplasty being performed on November 5, 1929. The cavity became apparently completely closed in ten, and was very small but not completely closed in three; one patient died. The anterolateral operations on the three patients whose cavities are not closed were performed early in the series before the importance of operating before new bone regenerated from the posterolateral periosteum was fully realized; in these three cases the anterior thoracoplasty was performed six months, six months and two months, respectively, after upper stage posterior thoracoplasty.

## SUMMARY

1. Nonclosure of large or stiff-walled cavities in the upper third of the lung is the most frequent cause of failure to effect cure of tuberculosis by artificial pneumothorax or surgical operation. Increasing experience with the surgery of pulmonary tuberculosis has demonstrated relatively safe means whereby the frequency of such failures may be greatly reduced.

2. Medium-sized and small cavities whose walls are not exceptionally rigid can usually be closed by an induced pneumothorax that is not necessarily complete, by phrenicectomy, extrapleural pneumolysis, paravertebral thoracoplasty of little or medium extensiveness, or by some combination of these or other less frequently used measures.

3. Large or stiff-walled cavities usually require for closure very extensive posterolateral thoracoplasty which for safety should be performed in three or more stages, and only rarely in two.

4. A special simple maneuver for gaining wide and easy access to the upper ribs, including the first, is described.

5. The value of posture, shot bags, an effective elastic brace and of preliminary parasternal chondrectomy in increasing the chance of complete closure of a cavity after thoracoplasty is discussed.

6. Incomplete closure of a cavity by a posterolateral thoracoplasty can usually be made complete by employing at the optimal time as a supplementary operation either anterolateral thoracoplasty, secondary posterolateral thoracoplasty or extrapleural pneumolysis with paraffin, pectoral muscles or rubber bag filling. Reasons are presented for preferring anterolateral thoracoplasty.

7. A simple technic is described for an anterolateral thoracoplasty that removes the upper cartilages and ribs, including the first, to the sternum and sometimes including part of the sternum.

8. Indications for anterolateral thoracoplasty to supplement extensive posterolateral thoracoplasty are relatively infrequent. Among approximately 100 patients who have had posterior thoracoplasty the combined operation was performed on 14 patients with large or unusually stiff-walled cavities. The cavities became apparently completely closed in 10, very small but not closed in 3; 1 patient died.

REFERENCES

1. Brunner, Alfred: Die chirurgische Behandlung der Lungentuberkulose, Leipzig, Johann Ambrosius Barth, 1924.
2. Sauerbruch, Ferdinand: Personal communication, July 23, 1930.
3. Sauerbruch, Ferdinand: Die Chirurgie der Brustorgane, ed. 3, Berlin, Julius Springer, 1930, vol. 1, pt. 2.
4. Brauer, Ludwig: Personal communication, August 29, 1929.
5. Hedblom, Carl: Anterolateral Costectomy for Inadequate Collapse Following Posterior Extrapleural Thoracoplasty, Arch. Surg. **21**: 1114, 1930.
6. Ziegler, Otto: Personal communication, July 29, 1930.
7. Proust and Maurer: La résection des apophyses transverses au cours de la thoracoplastie et l'apicolyse, Bull. et mém. de la Soc. nat. de Chir. **57**: 1292, 1931.
8. Lilienthal, Howard: Thoracic Surgery, Philadelphia and London, W. B. Saunders Co., 1925, vol. ii.
9. Balado, Manuel: La toracoplastia extrapleural, La Prensa méd., Argentina **9**: 672, 1922; Coffey, J. R.: Treatment of Surgical Collapse of the Lung, Ann. Surg. **86**: 683, 1927; Duval, Pierre, Quenu, Jean, and Welti, Henri: La thoracoplastie extrapleurale par voie axillaire: technique operatoire, Jour. de Chir. **32**: 641, December, 1928; Graf, Walther: Zur Resektion der ganzen ersten Rippe bei der extrapleuralen Thorakoplastik und über die Möglichkeit einer isolierten Entrippung von Spitzen—und Oberfelt (''thorakaplasticher Selektivkollaps'') Deutsche med. Wchnschr. **56**: 1647, September 26, 1930; Haugseth, Kristian: Fire tilfaelder av extrapleural thorakoplastik ved lungetuberkulose, Nosk. Mag. f. Laegevidensk **85**: 29, 1924; Jacobovici: La technique de la phrénicectomie et de la resection de la première côte dans le traitement chirurgical de la tuberculose pulmonaire, Bull. et mém. de la Soc. nat. de Chir. **52**: 994, November, 1926; Mallet, Guy, and Desjacques, P.: Technique de la resection des deux premières côtes par la voie postero-externe, sus-scapulaire, Lyon chir. **24**: 193, 1927; Piotet, Gustave, and Urech, Eugène: La thoracoplastie de Roux, Schweiz. med. Wchnschr. **10**: 602, 1929.
10. Brauer, Ludwig: Discussion, Ziegler, Otto: Bekämpfung der Lungentuberkulose durch Ruhigstellung der Lunge, Ztschr. f. Tuberk. **40**: 485, 1924.
11. Brauer, Ludwig: Das Ziel und die Abarten der extrapleuralen Thorakoplastik, sowie die Methodik der subscapular-paravertebralen Form, Beitr. z. Klin. d. Tuberk. **51**: 319, 1922.
12. Gale, Joseph: Personal communication, December, 1931.
13. Eden, Rudolph, and Robinson, Samuel: Versuche zur Lungenkollapstherapie, Beitr. z. Klin. d. Tuberk. **14**: 375, 1909.
14. Wilms, Max: Eine neue Methode zur Verengerung des Thorax bei Lungentuberkulose, München. med. Wchnschr. **58**: 777, 1911.
15. Woodruff, Warriner: A Simple Thoracoplasty Brace, Jour. Thor. Surg. **2**: 99, 1932.
16. Head, Jerome R.: Prevention of Regeneration of the Ribs, Arch. Surg. **14**: 1209, 1927.
17. Welles, E. S.: Accessory Thoracoplastic Operation for Collapse of Large Tuberculous Cavities, Arch. Surg. **14**: 384, January, 1927.
18. Gravesen, Johannes: Discussion of the Present Position of the Surgical Treatment of Pulmonary Tuberculosis, Brit. Med. Jour. **2**: 506, 1923.
19. Brunner, Alfred: Anzeigen und Ergebnisse der operativen Behandlung der Lungentuberkulose, Ergebn. d. inn. Med. u. Kinderh. **28**: 390, 1925.

20. Bérard, L., and Dumarest, F.: La thoracoplastie extra pleurale dans la traitement de la tuberculose pulmonaire, Arch. franco-belges de Chir. **26**: 423, 1923.
21. Alexander, John: The Surgery of Pulmonary Tuberculosis, Philadelphia, Lea & Febiger, 1925.

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## OPENING DISCUSSION OF SYMPOSIUM ON TUBERCULOSIS

PRESIDENT LORD.—It is evident from the morning demonstrations and the afternoon presentations that remarkable progress has been made in the collapse therapy of tuberculosis. It is, however, unfortunate that the adoption of this method of collapse therapy throughout the country has not kept pace with its advantages.

The matter is now open for discussion.

## DISCUSSION ON TUESDAY AFTERNOON'S SYMPOSIUM ON TUBERCULOSIS

DR. EDWARD W. ARCHIBALD (MONTREAL, CANADA).—I do not think that a symposium of this sort should be allowed to terminate with absolutely no discussion.

As Dr. Lord has said, it has given us evidence of the recent enormous extension of collapse therapy. Fifteen years ago or so we used to think that possibly one or two per cent of tuberculous patients might be candidates for collapse therapy; today we have been told that 75 per cent come within that range. Of course, one must remember that, in any such estimation, pneumothorax is counted as one form of collapse therapy, and rightly enough; but as a surgical society we have not been used to considering pneumothorax as a surgical measure.

What strikes me most in today's addresses is the apparent enormous increase of the general confidence in the value of minor operations, chiefly phrenic exairesis, and the relative retrogression of the idea of thoracoplasty. I am not at all sure that that swing of the pendulum is altogether a good thing. Indeed, I suspect that, within the lapse of a few years, it will be found that phrenic exairesis, and various other minor operations, which are now being used so extensively as an independent, and often the sole operation, will end up by giving us imperfect results, and often disastrous results. The frequently imperfect effect of phrenic exairesis in the long run is to be set against the immediate mortality of thoracoplasty in the short run; and I think that the comparison, in respect of apparently permanent arrest and restoration of work capacity, will be found to be greatly in favor of thoracoplasty, whereas the operative mortality of thoracoplasty is now brought down to a very low figure in properly selected cases.

A phrenicectomy, performed as an independent collapse operation, frequently improves symptoms to such an extent that the patient, and also the patient's doctor, remains satisfied with a partial result and refuses the proper and thorough collapse operation of thoracoplasty. But the disease is only partially held, and, after a variable delay, there appears an extension of the disease in the good lung, which now makes thoracoplasty impossible.

I have no intention of denying the good effects of phrenic exairesis, in many patients. It is perfectly correct also to say that quite a few of these patients have made such recoveries that they apparently no longer needed a thoracoplasty. But we must remember that much of this recent enthusiasm over apparent permanent arrest is derived from an experience of very recent years. I still believe that many a patient who has had a phrenicectomy done, and nothing more because he was "so much better," is going to die within five years, when if he had had a thoracoplasty at the proper time he might not have died. Thoracoplasty will more and more return to favor, in the measure in which we learn to select cases safely for it.

If I may impose on your patience one or two minutes more, I should like to say a word or two concerning the cases of mixed infection empyema, referring particularly to Dr. Berry's most interesting paper.

I shall make only one point, which is this: that I suspect that many people still think of thoracoplasty as the proper procedure to be adopted whenever there is a mixed infection empyema, as if the thoracoplasty of itself cured the disease. I have been asked on several occasions to do a thoracoplasty in the very flush of a most violent infection. This is wrong. Let us consider the subject generally, for a moment. In the question of tuberculosis I have always held that the surgeon must think *fundamentally*. Of course, it is true that he has to think also *mechanically* in terms of compression or collapse, but we must not forget to think, first of all, in terms of *resistance*. When I say fundamentally, I mean resistance. Whatever that thing resistance is due to, or consists of, we all recognize it clinically. Now, when a patient has developed an empyema containing staphylococci, streptococci, possibly anaerobes, as well as tubercle bacilli, and is running high fever, I quite agree with Dr. Berry that one should first spend some time in a trial of aspirations and irrigations. I have had a certain small measure of success, in an experience which is not quite as large as his, with such procedures, and when such measures do succeed, it is clear that the patient possesses resistance both to the tubercle bacillus and to the mixed infection. Such patients rarely die of their empyema.

But I also agree with him that one must not wait very long if fever and a rapid pulse continue. If the patient is obviously slipping, one should not delay instituting proper drainage. What next? Of two things, one: the drainage either relieves the infection, or it does not. We all know that even after thoroughly good drainage has been instituted the pulse and temperature may remain up, and death may ensue inside a few months. Such patients obviously have no resistance. They simply succumb to their infection. Others are extraordinarily benefited by drainage, and gradually climb to a satisfactory level of general health.

At this stage comes the point which I think it is necessary to emphasize. It is only after it has been established by the good effects of drainage that the patient possesses resistance, as shown in normal temperature, normal pulse, gain in weight, and so on; and only after months of that improved condition that one should undertake thoracoplasty. Thoracoplasty undertaken as a measure to overcome the infection of these grave empyemas is inadmissible; it is a *non sequitur*—it simply doesn't apply. Pus has to be evacuated, not put under compression or tension. Yet I find, after talking with some men who, though interested in tuberculosis and in thoracic surgery, have not thought deeply enough about it, that there is a sort of rule of thumb idea prevalent to the effect that thoracoplasty on such patients is indicated at any stage of the infection. No! Thoracoplasty, in the treatment of tuberculous empyema is of value only in the case of patients who are good chronics and for the purpose of obliterating a long-standing discharging pleural cavity. That cavity must be allowed to lose its infectivity before one undertakes thoracoplasty.

Open drainage is the test by which one can weed out the good cases from the bad, those who have resistance from those who have it not. After drainage, one must wait from three months to six months, or more; and then in the selected cases, one can get quite good results from the operation of thoracoplasty.

I haven't gone over my figures lately, but I did so about four years ago, and I found I had got out of seven cases at that time practically a cure in four, two of whom could work and were working, and two of whom could work but didn't want to. Only one died. One was in the *statu quo ante*, while the other was improving. I welcome this excellent analysis of Dr. Berry's of such a large number of cases, as I am confident that it will be of much real help to us all, a valuable guide in a bit of country where the going is particularly bad. I should have liked to discuss Dr.

Hedblom's splendid paper, based as it is on so wide an experience; but the hour is too late.

If I have talked too long, I beg your pardon. The whole symposium, covering so great a territory, has left my head in a bit of a whirl. You will, however, allow me one final remark. I would like to say, as coming from the north of the line, how much I admire, how greatly I wonder at this extraordinary—well, the French word is *essor*, let us say, this uprush, this inspired and inspiring upswing of thoracic surgery during the last few years, for which America, and in particular this Society has been responsible. (Applause.)

# THE MECHANISM OF THE DEVELOPMENT OF TUBERCULOUS PNEUMONIA FOLLOWING THORACOPLASTY*

HOWARD A. MCCORDOCK, M.D., AND HARRY BALLON, M.D.
ST. LOUIS, MO.

WE HAVE concerned ourselves with the mechanism underlying the development of tuberculous pneumonia following thoracoplasty; for in the past relatively little attention has been paid to it in connection with surgical procedures upon tuberculous lungs. Clinical discussions regarding thoracoplasty have emphasized the relative importance of removing either the upper ribs or the lower ribs first, and have attempted to explain the pneumonic reaction from this point of view. We question whether the mechanism of tuberculous pneumonia following thoracoplasty is dependent merely upon the order of the operation performed, since many thoracoplasty operations have been performed both ways, and particularly since they have been preceded frequently by phrenicectomy. The reported unfavorable results have been practically the same, although delayed occasionally when the lower ribs were removed first. Recorded experiences would tend to show, and rightly so, that the amount of compression which has been obtained and the location of this compression play a part in the subsequent development of tuberculous pneumonia. One consideration is obviously whether or not the tuberculous pneumonia, which develops in the so-called good lung, is the result of a spreading of the tuberculous process from a focus in this lung which has become activated, or whether it is an extension from the affected lung which is being subjected to compression therapy. Quite commonly those deaths which occur within the first few days following thoracoplasty are considered as being due to an edema of the lungs or to a bronchopneumonia of a nontuberculous nature. If the patient survives for two or more weeks the diagnosis of tuberculous pneumonia is then usually made. This erroneous interpretation is probably due to the failure on the part of the clinicians and pathologists to appreciate the fact that the inflammatory edema is merely an early manifestation of and part of a tuberculous pneumonia. We have had the opportunity of examining the lungs of patients who died from pulmonary complications following extrapleural thoracoplasty for pulmonary tuberculosis. Our findings in two cases only have been recorded and are as follows:

CASE 1.—M. C. W., 29966, female, aged twenty-nine, was first admitted to Barnes Hospital on June 8, 1931. Her complaints were productive cough, occasionally blood streaked, and pain in the chest; all of two years' duration. In her past his-

*From the Departments of Pathology and Surgery of Washington University School of Medicine, St. Louis, Mo.
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tory mention is made of an appendectomy in 1927, but without any description being given of the pathologic findings.

*History of Present Illness.*—The onset of the patient's condition was rather insidious. It apparently developed following a primary pleurisy. One year after the onset of her illness she went to a sanitorium in Arizona where she received two pneumothorax treatments every week for one year. She responded moderately well to this treatment. During the entire two-year period until just previous to admission she maintained her usual weight which was 110 pounds. During the week preceding her entrance into the hospital, however, she lost four pounds in weight. Since that time she noticed also that she was getting hoarse.

*Physical Findings.*—The physical examination and roentgen ray findings suggested the presence of a cavity in the left upper lobe (Fig. 1). Her blood pressure was 110/90, her vital capacity on June 10, 1931, was only 1000 c.c. Examination of her

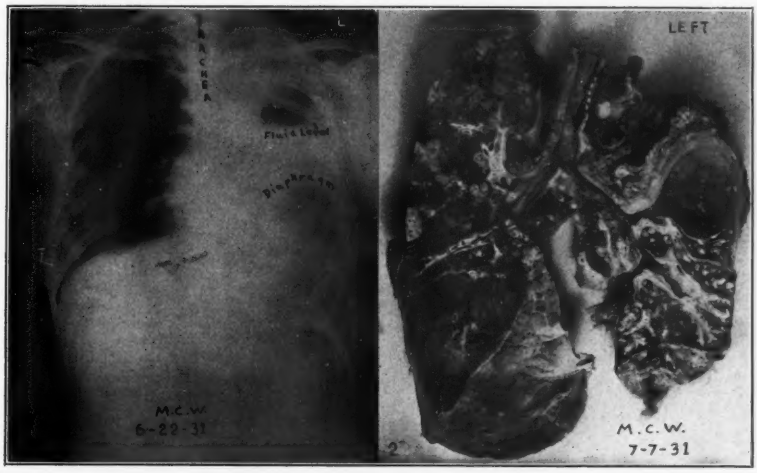


Fig. 1.—(Case 1.) *1.* This film (6/22/31) taken following the phrenicectomy (6/13/31) corresponds in every respect with a film taken just previous to the performance of the thoracoplasty (7/3/31). Note the midline position of the trachea and the fluid level in the pulmonary cavity.
*2.* Shows the large apical cavity in the left lung and the extensive involvement of the right lung by tuberculous pneumonia. The right lower lobe is comparatively free.

larynx at this time showed no important pathologic changes. It was therefore decided to subject the patient to a phrenicectomy, which was done by Dr. Graham on June 13, 1931, when five inches of the phrenic nerve were obtained. The patient showed no ill effects from this procedure. The diaphragm rose slightly and her cough and expectoration were somewhat diminished. Her sputum still showed many acid-fast bacilli and her white blood cells on June 22, 1931, numbered 6,200, the differential count showing 2 eosinophils, 2 basophils, 3 stabs, 66 segmented cells, 1 monocyte and 26 lymphocytes. The red blood cells numbered 4,090,000, the amount of hemoglobin was diminished to 73 per cent. About three weeks after the phrenicectomy the patient began to have gastrointestinal symptoms. She was given a swallow of barium but nothing unusual was observed in the esophagus. A gastrointestinal examination gave indefinite results. Her larynx was again examined on July 23, and seemed to be within the range of normal except for considerable redness in the interarytenoid area. The patient was discharged on Sepetmber 2, 1931, be-

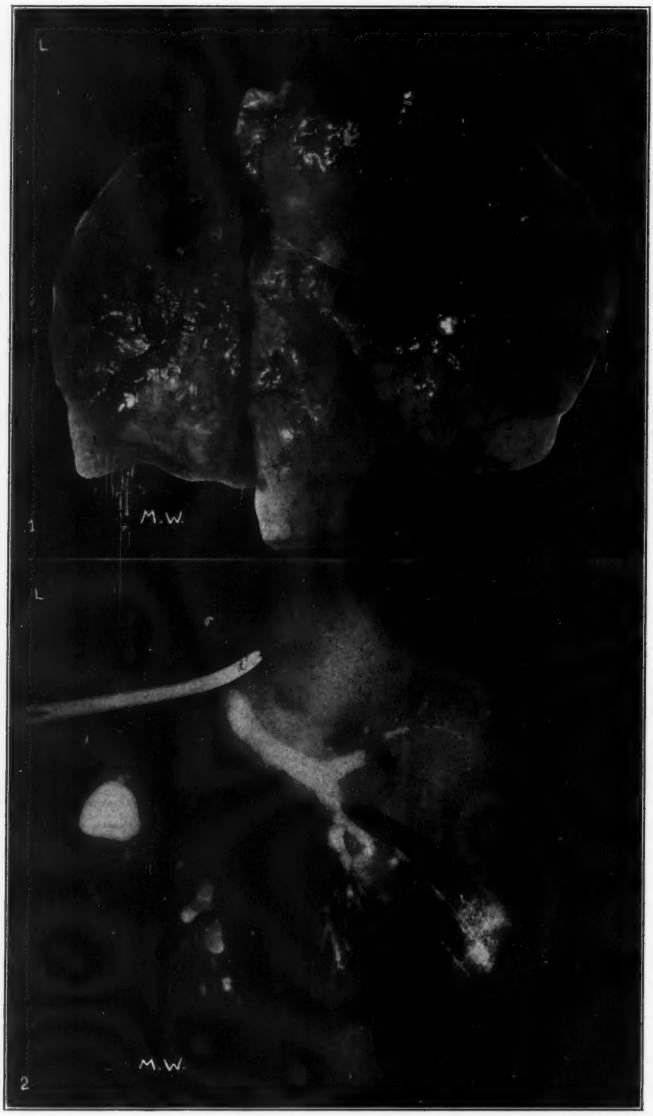


Fig. 2.—(Case 1.) *1.* (Posterior view.) Note the small left lung and the large right lung. Part of the increase in the size of the right lung is undoubtedly due to the pneumonic process, but most of it is probably due to compensatory emphysema. The left lung is small because of the compression which was obtained by the phrenicectomy (basal) and by the partial thoracoplasty, (apical and lateral).

*2.* Represents the roentgen-ray appearance of both lungs, (necropsy specimen) following the injection of lipiodol (posterior view). Note that practically the only remaining portion of air-containing lung tissue is in the right lower lobe. The remaining portions of both lungs present the appearance of consolidation. Some lipiodol has entered the cavity in the left upper lobe. The lipiodol failed to enter the right middle and upper lobes because of the pneumonic process.

cause of the death of her mother. She was requested to return at a future date for a thoracoplasty.

She was readmitted on September 19, 1931. The interval between the first and second admission was uneventful and the physical findings showed no change. The patient's vital capacity on September 24, was 1200 c.c. Examination of the larynx on September 28, 1931, suggested an early tuberculous laryngitis. The patient's

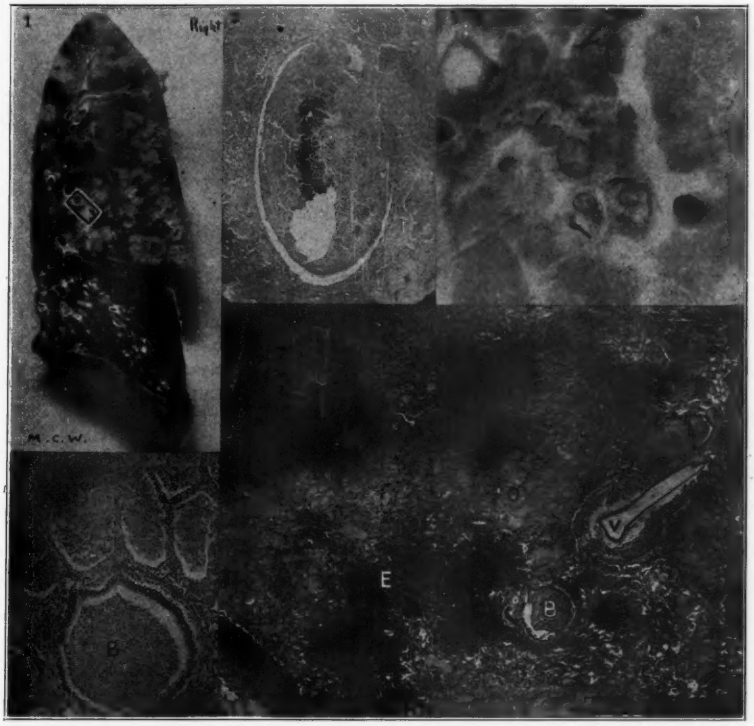


Fig. 3.—*1.* Represents a cut section of the right lung of the patient referred to in Case 1. Two bronchi filled with gelatinous material, which contained many tubercle bacilli, are enclosed in the rectangle.

*2.* Represents a photomicrograph of a smaller bronchus similar to the ones indicated in the rectangle in Fig. 1. The dark material represents clumps of tubercle bacilli.

*3.* Cellular exudate from an alveolus in an area of pneumonia showing tubercle bacilli within large mononuclear cells.

*4.* Low power photomicrograph of a section from the right lung showing areas of edema (light staining, and indicated by letter O) and areas of cellular exudate (dark staining, and indicated by letter E). B = Bronchus. V = Vein.

*5.* High power photomicrograph showing the characteristic cellular exudate of the tuberculous pneumonia in a bronchus and a few surrounding alveoli. This exudate is composed chiefly of large mononuclear cells, lymphocytes and coagulated fluid. The only microorganisms observed in this exudate were tubercle bacilli. (B = Bronchus.)

resting pulse was 128, immediately after sitting and lying down ten times it was 132, and one minute later, 124. A roentgen ray film at this time still showed slight displacement of the mediastinal contents to the affected side. It was fully appreciated that this patient was a poor surgical risk. Since the lesion was unilateral, a thoracoplasty was recommended and portions of the 5th, 4th, 3rd, 2nd and 1st ribs

were resected by Dr. Graham on October 3, 1931. Her pulse before operation was always between 110 and 130. Immediately following the operation it rose to 140. Thirty-six hours after the operation she became very dyspneic, slightly cyanotic and complained of pain on respiration. Her temperature was 39.4° C. She was placed in an oxygen tent, but died on October 7, 1931, from what was clinically considered to be bronchopneumonia. A necropsy was performed by Dr. E. W. Thurston.

*Necropsy Findings.—Right Lung.*—The upper lobe of the right lung was solid. Through the thin unaltered pleura one could see gray consolidated lobules and boggy edematous intervening lung tissue. Small solid areas could be felt in the middle and lower lobes, but the tissue was generally crepitant. On section, the upper lobe presented the appearance of a typical early tuberculous pneumonia, bronchial in distribution. Lobules of gray consolidation were scattered upon the surface and between these the alveoli were filled with a viscid fluid producing a gelatinous appearance in the remaining lung tissue.

A few tiny gray areas were seen in the middle lobe but none in the lower lobe. Both the lower and middle lobes were air-containing except for a small area at the base which showed some edema.

*Left Lung.*—The collapsed left lung was about two-thirds its normal size, and was covered by a thickened pleura (Fig. 2). A large cavity occupying the greater part of the upper lobe was discovered after sectioning the lung. This cavity was lined with a thick layer of soft yellow material and contained a small quantity of thick yellow fluid, smears of which revealed many tubercle bacilli.

The fissure between the lobes was obliterated by fibrous tissue. No cavities were discovered in the lower lobe. Plugs of thick yellowish material, similar to that found in the cavity filled many of the smaller bronchi, and in places masses of it were adherent to the wall of the main bronchus. Small caseous patches were scattered throughout the parenchyma of the lower lobe. Between these patches the alveoli were collapsed and the tissue was airless.

Many calcified nodules were found in the bronchopulmonary lymph nodes. Both upper and lower tracheo-bronchial groups of lymph nodes were greatly enlarged but contained caseous foci only and no calcified tubercles.

*Microscopic Examination.—Right Lung.*—In the sections from the right upper lobe there were many areas of consolidation, lobular in distribution, in which the exudate was composed principally of large mononuclear cells with a few polymorphonuclear (Fig. 3). Foci of necrosis were found in this exudate such as are commonly seen in the exudative type of tuberculous pneumonia. Plugs of necrotic exudate filled many of the smaller bronchi. Sections stained for acid-fast bacteria revealed the presence of moderate numbers of tubercle bacilli, which were usually situated within large mononuclear cells. One bronchus contained large clumps of tubercle bacilli in the necrotic exudate that filled its lumen. These clumps were similar to those seen in the lining of the cavity in the opposite lung. Between these areas of cellular consolidation the alveoli were filled with pink staining coagulated albuminous fluid containing a few cells and occasionally a little fibrin. This is the material that imparts the gelatinous character to the tuberculous pneumonia. In this gelatinous exudate, an occasional cell containing a phagocytized bacillus could be found.

*Left Lung.*—The wall of the cavity in the left upper lobe was composed of dense fibrous tissue and was lined with a thick layer of necrotic debris. Clumps of tubercle bacilli could be seen with acid-fast stains in this necrotic layer. The lower lobe showed collapsed alveoli, bronchi plugged with caseous material and old fibrocaseous foci.

The lymph nodes from the left side contained a few old calcified tubercles and many fibrocaseous ones. Those on the right side showed no trace of old lesions, but only a few fresh cellular tubercles.

*Discussion.*—In this case it could be clearly demonstrated that the lesion in the so-called good lung (right) was an extension from the cavity in the left lung, which was lined with material rich in tubercle bacilli and which had been subjected to compression therapy. Tubercle bacilli could be demonstrated in large numbers in the necrotic material lining the cavity and were also observed in the corresponding bronchus. The opposite lung, except the lower lobe, showed foci of tuberculous pneumonia which were surrounded by areas of edema. This edema was situated in the alveolar spaces and contained mononuclear cells, some of which had phagocytosed an occasional tubercle bacillus. The small bronchi in the centers of these pneumonic patches often contained plugs of material similar to that found in the tuberculous cavity which had been subjected to compression. Large masses of tubercle bacilli could be demonstrated in these smaller bronchi. The patient referred to in this case was a poor surgical risk, but there was no clinical or roentgen ray evidence of the presence of an active tuberculous focus in the so-called good lung. This patient died five days after a partial thoracoplasty and judging from experimental observations (Rich and McCordock)[1] the pneumonia in the so-called good lung need not be of more than five days' duration. The presence of tubercle bacilli in the latter could not be attributed to a preformed focus in this lung, because there was no lesion older than the pneumonic process that could have supplied the bacilli necessary to induce the pneumonia.

CASE 2.—M. M. G., 29470, female, aged twenty-four, was first admitted to Barnes Hospital on May 5, 1931. Her complaints were cough, expectoration and occasional hemoptysis, all of five years' duration. During this period the only treatment had been absolute rest in bed for 2½ years previous to admission. This had resulted in a gain of 24 pounds in weight, but during this period she also suffered from dyspnea on exertion. She was never subjected to pneumothorax treatment and when first observed at the Barnes Hospital the duration of her illness and the nature of the lesion did not warrant attempting it. Her past history was negative.

The positive findings were entirely referable to the right chest. The process was one of cavitation (Fig. 4). There were no abnormal findings in the contralateral lung. The heart was centrally placed and shifted somewhat with change in position and with respiration. The blood count on May 5, 1931, showed 17,600 leucocytes. Schilling differential count showed 1 eosinophil, 10 stabs, 73 segmented cells, 9 lymphocytes and 7 monocytes.

Phrenicectomy was performed by Dr. Brock on May 9, 1931. The entire phrenic nerve was obtained. The diaphragm rose slightly following this procedure and exhibited paradox movement. A thoracoplasty was considered about this time (June 9, 1931). It was not undertaken because of a high stab count and recent gastric disturbances. The midline position of the trachea, and the fact that the heart shifted with change of position were other considerations making this operation inadvisable. She was therefore discharged to her home on June 12 to continue her rest cure. She was readmitted on September 28 of the same year. During the interval the patient enjoyed relatively good health. She was up and about for approximately five hours every day. This amount of exercise did not seem to tire her in any way. Her vital capacity on September 21, 1931, was 1200 c.c. Examination of her

larynx at this time showed no evidence of tuberculosis. Her pulse rate on October 1, 1931, was 92 after sitting and lying down ten times, one minute later it was 104, two minutes later 92. Her mediastinal contents were still in the midline, however. Nevertheless, since the lesion was unilateral and since the only possible way to effect a cure was by thoracoplasty this was undertaken by Dr. Graham on October 5, 1931, when portions of the 11th, 10th, 9th and 8th ribs were resected. For the first twenty-four hours the patient's postoperative condition was satisfactory, then she became cyanosed and dyspneic and required oxygen. She remained in an oxygen tent until the time of her death which was on October 23, 1931, and apparently due to a bronchopneumonia which involved the greater part of both lungs. A necropsy was performed by Dr. Elizabeth Moore.

*Necropsy Findings.—Right Lung.*—The right lung contained a large cavity posteriorly, extending from the base to a point within an inch or two of the apex.

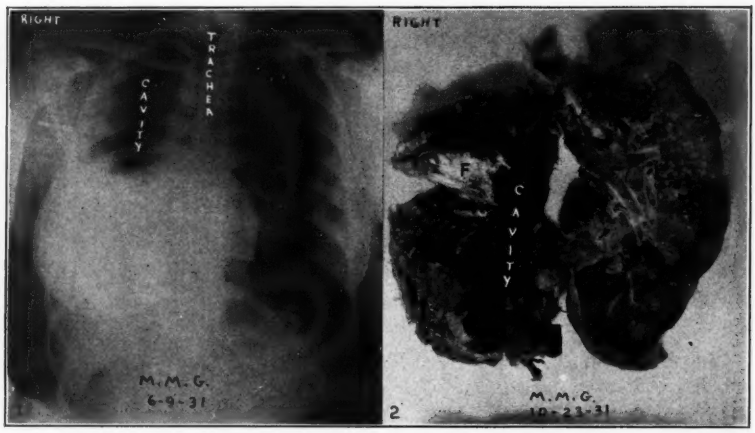


Fig. 4.—*1*, is the roentgen-ray appearance of the chest of patient referred to in Case 2. This film was taken on 6/9/31. The phrenicectomy was performed on 5/9/31, the thoracoplasty 10/5/31. A film taken previous to the final operation is similar in every respect to the above. The trachea is slightly displaced to the right. The large pulmonary cavity acted like a veritable pneumothorax pocket and really tended to displace the trachea. The right diaphragm cannot be visualized. The left lung appears to be clear.

2. *F*—large softened caseous area in the right upper lobe which communicated with a bronchus. The rest of the right lung is merely one large cavity. The left lung shows foci of tuberculous bronchopneumonia in both lobes.

Thickened pleura formed the posterior wall of the cavity. Granulation tissue lined the cavity, but in places this lining was interspersed with small areas of necrotic tissue covered with fibrin.

The entire apical portion of this lung was composed of soft, caseous material, that in places was beginning to break down and liquefy. The bronchi of the upper and middle lobes were filled with soft, caseous necrotic plugs. In the lower lobe there was a great deal of scarring in addition to some old fibrous, partly calcified foci. Calcified tubercles were found in the tracheo-bronchial nodes on this side.

*Left Lung.*—The left lung showed a widespread tuberculous pneumonia with large confluent areas in the lower lobe and scattered foci about bronchi in the upper lobe. In most places the exudate was yellow and opaque, presenting the appearance of beginning necrosis and caseation. The gelatinous exudate and the edema seen in the preceding case of M. W. were not a conspicuous feature here. The lymph nodes

at the hilus showed only a few tiny tubercles which were distinctly smaller than those on the opposite side.

No old focus could be found in the left lung.

*Microscopic Examination.—Right Lung.*—Sections from the wall of the cavity showed it to be lined with granulation tissue in which no bacilli could be found. The apex of this lung no longer contained any recognizable pulmonary structures, but was reduced to a mass of caseous debris sprinkled in places with a fine dustlike deposit of calcium. Bacilli could be found by means of acid-fast stains, although they were not present in the enormous numbers seen in the first case.

*Left Lung.*—The left lung showed a tuberculous pneumonia in which the cellular exudate was necrotic in the majority of the sections. An occasional tubercle bacillus could be seen, situated usually within a large mononuclear cell. Some of the alveoli were filled with coagulated albuminous fluid, especially in the lower lobe. The pneumonia in this case was also bronchiogenic and had not been produced by blood stream dissemination.

The source of the bacilli responsible for the pneumonia in the contralateral lung was not as obvious as in the first case. Of course it could not have been the rather clean cavity. The oldest softened caseous focus in the apex of the right lung was the only possible source of the large numbers of bacilli or of tuberculo-protein derived from disintegrated bacilli.

Tuberculous pneumonia of about the same stage as that seen in this lung has been produced in sensitized animals in ten days, and in the case here described, 18 days had elapsed between operation and death.

## DISCUSSION

The patient in the second case died 18 days after the performance of a partial thoracoplasty. The mechanism, which was responsible for death in this case, was practically the same as in the first one, although the large cavity, which was present in her originally involved lung, did not contain many tubercle bacilli. However, an active softened caseous focus was present in the apex of that lung which communicated with a bronchus. This is regarded by us as the source of the tubercle bacilli, or their products which caused the tuberculous pneumonia in the opposite lung. The absence of generalized edema in both lungs rules out the possibility of cardiac failure. The absence of other bacteria in the pneumonic areas renders the possibility of a nontuberculous pneumonia unlikely.

## SUMMARY

In the light of the above and other similar but unrecorded experiences, the suggestion seems justified that many of the so-called pneumonias and edemas, which develop following thoracoplasty in cases of pulmonary tuberculosis, are of a tuberculous nature. That these tuberculous lesions are not the result of a hematogenous dissemination can be readily shown by the absence of tubercles in other organs. Perhaps the fact that some patients recover from these induced tuberculous pneumonias is due to the relative paucity of tubercle bacilli in these lesions. This view is supported by the experiments of Austrian and Willis,[2] who have shown that the presence of dead or disintegrated bacteria and their products

may result in a tuberculin reaction which can, of course, resolve completely, or proceed to the formation of hard tubercles. One might ask why tuberculous pneumonia develops following the operation, when lesions fail to develop in the good lung before the operation, despite the presence of tubercle bacilli-laden sputum, which must bathe the tracheobronchial tree? The past work of Rich and McCordock,[1] we believe, supplies a possible answer; they have shown that the important consideration is the number of tubercle bacilli that reach the alveoli. Patients subjected to thoracoplasty are all potentially sensitized individuals, and the compression therapy merely acts as an inoculating mechanism, which leads to the sudden discharge of relatively large quantities of tubercle bacilli or their products into the alveoli of uninvolved lung tissue. The above would suggest that preoperative prophylactic measures of a suitable kind might prevent the occurrence of a secondary tuberculous pneumonia in the previously nonaffected lung, or at least lessen its frequency.

REFERENCES

1. Rich, A. R., and McCordock, H. A.: An Enquiry Concerning the Rôle of Allergy, Immunity and Other Factors of Importance in the Pathogenesis of Human Tuberculosis, Bull. Johns Hopkins Hosp. **44**: 273, May, 1929.
2. Austrian, C. R., and Willis, H. S.: The Pulmonary Effect of Intratracheal Injections of Old Tuberculin in Rabbits, Am. Rev. Tuberc. **23**: 310, March, 1931.

DISCUSSION

DR. EVARTS A. GRAHAM (ST. LOUIS).—This study that Dr. Ballon and Dr. McCordock have made of these two fatal cases has interested me tremendously. I think that in retrospect it shows that, particularly in the first case, I made a mistake in not draining the cavity bronchoscopically before the operation. This woman had a large cavity which, at the time of the operation, so far as we knew contained fluid. The possibility of causing damage in the other lung by squeezing all this tuberculin and living tubercle bacilli into it, I confess had not seriously entered my head. I believe, however, if this patient had had bronchoscopic drainage immediately before the operation in order to get rid of all the detritus-containing tuberculin as well as living bacilli, perhaps she would not have developed this rapidly fulminating pulmonary edema which caused her death in five days. The edema seems to me to have been undoubtedly due very largely to a tuberculinization of the other lung. This matter also I think is of interest in connection with the paper presented by Dr. Coryllos on Monday, suggesting that continuous suction be provided through an intratracheal catheter during the performance of the thoracoplasty.

This report is merely a preliminary one. I think it is always unwise and dangerous to make generalizations from two cases, but it seems to me that it does bring up an important point, one which we shall have to consider more fully in performing thoracoplasties and other compressive procedures on patients who particularly have large cavities.

DR. EDWARD W. ARCHIBALD (MONTREAL, CANADA).—Dr. Ballon's paper it seems to me, is a very important one. The problem of how to escape such unfortunate spreads to the good lung as a result of operation has exercised our minds a great deal. Fortunately, the occurrence is not a very frequent one, and still less frequently fatal.

At one time, some years ago, in going over my figures, I found we had 3 per cent of deaths following thoracoplasty because of this complication.

Some years ago, Dr. Lincoln Brown, now of San Francisco, who was working in our laboratory, showed in a very simple way that if the sputum is fluid there is danger of spread to the other side, but that with the ordinary mucopurulent tenacious sputum, such extension does not occur. He injected lipiodol into sputum, of both types, and demonstrated this fact in animals quite clearly by x-ray films.

As a matter of fact, that is what one would expect. If sputum is tenacious, it cannot penetrate into the bronchi and is inevitably coughed up in no long delay. In this case, apparently the sputum was rather fluid. Whether one could have suctioned it off in any way before operation, I doubt. Some sputum, it is true, is evacuated by posture, or by bronchoscope, but there is always some left behind. I prefer to overcome or to guard myself against this danger by the posture of the patient on the operating table, which is in the horizontal position, with the chest elevated on a pillow; and consequently with the head lower than the bifurcation of the trachea.

For my own part, I cannot think that the explanation that Dr. Ballon gives of actual-contact infection accounts for all the cases. We find a flare-up on the good side occurring in practically dry cases. We find it also occurring and clearing up inside of a month or two, which seems to me almost irreconcilable with acute tuberculous infection—not quite, of course. So I have come to think that a certain number of such cases must represent an allergic phenomenon if that theory is still accepted by the more scientific people. Old organic lesions, sometimes undiscoverable by the ordinary x-ray film, are aroused in this condition to simulate a bronchopneumonia, by what amounts to a tuberculin injection brought about by the compression effects of the thoracoplasty. That seems to me the most likely explanation to account for those patients who recover promptly, surprisingly so, after what looks like a threatening tuberculous bronchopneumonia.

We must all be grateful to Dr. Ballon for giving us this clear report of a thorough pathologic examination of the lesions of acute postoperative infection in the good lung, an opportunity of which most of us surgeons have taken too little advantage hitherto.

DR. DEAN B. COLE (RICHMOND, VIRGINIA).—I wish to call attention to the use of postural drainage prior to the surgical collapse of pulmonary cavities. Most patients are accustomed to draining their cavities daily and know pretty well when the cavity is empty. We employ postural drainage routinely in these patients prior to operation, and if the patient states the cavity has not emptied, the operation is postponed until emptying occurs.

DR. HARRY BALLON (ST. LOUIS.)—I merely wish to reiterate that our presentation at the moment is concerned with two cases. As Dr. Archibald pointed out, it is likely that the mechanism to which we refer does not operate in so-called "dry" cases—cases in which there is no fluid in the cavity. As a matter of fact, we lean towards his suggestion that the reaction in "dry" cases is probably allergic.

Several years ago I made some observations upon the lipolytic activity of the blood before and after compression therapy including thoracoplasty and found a distinct rise after compression therapy. This rise was interpreted in terms of a tuberculin reaction. In other words, as a result of compression "tuberculin-like" material had been introduced into the circulation.

We would also draw attention to the fact that a large percentage of the immediate deaths following thoracoplasty for pulmonary tuberculosis are recorded as being due merely to edema of the lungs.

# DANGERS AND COMPLICATIONS OF OLEOTHORAX*

## Report of Cases

J. N. Hayes, M.D.
Saranac Lake, N. Y.

WITH the increasing use of oleothorax in pulmonary collapse therapy, it is important to know the accidents which may accompany it. Of 150 cases reported by Marie and by Fontaine, 39 deaths resulted, some of which were directly attributable to the method. Anyone employing this valuable treatment should make himself thoroughly familiar with papers of the French school, especially those of Bernou, Küss and Fontaine. Küss blames most of the errors on improper technic.

Oil, especially when cold, is irritating to the pleura of many patients. It is, therefore, advisable to inject it when warmed to 38° C. in a water-bath. This also facilitates an easy flow. The first injection should be about 5 c.c. to test the reaction of the pleura. This should always be done if the pleura is dry, but is not necessary when pus has previously been aspirated. If gomenol is to be incorporated with the oil it is preferable to use oil without gomenol for the test dose. Frequently the pleura is sensitive, and fever, usually slight and of short duration, occurs. It rises to 100° or 102° and falls within a few days. Slight soreness in the chest may accompany it. The fever may last for a week or two, however, and no further injection should be made until the fever falls. On the next injection, of perhaps 10 c.c., another febrile reaction may occur but usually as one persists in the treatments the reactions cease. But if they continue it is recommended that one change the character of the oil; e. g., from paraffin to olive. It may be possible to continue thenceforth without febrile reactions. Or it may be necessary to give up the treatment on account of persistent reactions. I find reactions to paraffin oil frequent and slight but have never had to substitute olive oil or to stop on account of reactions.

Febrile reactions are frequently followed by serous effusion, usually small in amount and of no importance in itself. It may be dangerous, however, when it causes the gas pressure to rise too high. While it is rarely necessary to aspirate the fluid, the patient must be watched carefully for signs of pressure (increased respiratory and cardiac rates) and the gas pressure lowered when indicated.

Case 1.—A patient, whose effective collapse was menaced by expansion following serous effusion and whose pleural cavity had now been dry for three months, was given 5 c.c. of pure warm paraffin oil on September 22. Temperature remained normal until September 25, when it was 101°; on the following two days it was 100°

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and then normal. No inconvenience otherwise. On September 29, 110 c.c. paraffin oil were injected with a final gas pressure of 0 to −2. This was followed by a severe febrile reaction (102° maximum, to 99° on October 12) and marked gastric disturbance. No symptoms referable to the chest. On October 14 the gas pressure was +13 to +12; this was lowered to +4 to +1 by withdrawing gas. The temperature was normal after October 15. An x-ray film showed fluid beneath the oil. On October 29, 350 c.c. of seropus were aspirated. The refills of oil were then continued.

On account of this possible rise in pressure, which may be sudden, the gas pressure at the beginning of oleothorax should be left no higher than neutral. As Fontaine remarks, these increases in pressure are more dangerous in small stiff-pleural pockets.

When serous effusion persists, increases and must be aspirated repeatedly, it may become puriform, although Fontaine says this is rare. Hemorrhagic pleurisy is also very uncommon.

After the initial, tentative injections in the case of a dry pleura, or early in the treatment when purulent effusion is present and larger amounts may be injected at one time, accidents of too high pressure may occur. The gas pressure, as long as gas is present, must be under constant observation during an injection. For theoretical safety two needles are advisable, one connected with the oil syringe, the other with a water manometer. As oil is injected an equal amount of air is withdrawn by means of a pneumothorax apparatus with the bottles in position for aspiration. In a large dry pleura in the early stages of installation of oleothorax when the gas pocket is relatively large, I use one needle attached by means of a three-way adapter to a 10 c.c. syringe. After each injection of 10 c.c. of oil, 10 c.c. of gas is discharged through the tubing leading from the adapter to the oil container. The syringe is then refilled with oil by means of the same tubing and the process is repeated until the desired amount, usually 100 to 200 c.c., has been injected. Frequent connections with a water manometer usually show no change in pressure, but, if the pressure rises slightly, more gas may be withdrawn. At this stage this method is easy, safe, and expeditious. If the gas pressure is not under observation and rises high, the symptoms are very characteristic: "sensation of malaise at first, followed by anguish with intense dyspnea and sensation of choking; it is really an accident of suffocation" (Fontaine). This is more likely to occur after a large amount of oil has been injected. At this point especially, fluoroscopic or radioscopic control is very necessary. One must determine how large is the remaining gas pocket and how best to approach it; i. e., in what position to place the patient, for the needle must enter the gas pocket. It is not necessary to fill completely the pleural space; a small bubble of gas, from which to obtain the final pressure, may be left to absorb. When the filling is nearly complete, it is imperative to control the gas pressure constantly and this is best done by using two needles placed near each other. If the pocket is filled completely, oil will escape from the "pres-

sure'' needle and may be observed in a glass tube inserted into the rubber tubing near the needle. If the pocket is to be completely filled, the last few centimeters of oil may elevate the oil pressure very considerably. When symptoms of overpressure occur, oil should be withdrawn immediately, the patency of the ''pressure'' needle in the gas pocket determined and gas withdrawn.

Perforation of the lung may occur during the treatment, either at the time of installation or in the intervals between injections. In Marie's 50 cases it occurred in 7 with death in 6; in Fontaine's 100 collected cases in 10 with 9 deaths. It may be due to faulty technic or to evolution of the disease within the lung. Since the use of oleothorax small perforations which had not been previously recognized during pneumothorax treatment have been detected with the beginning of oleothorax. These are not due to the latter but are revealed by the presence of a few drops of oil in the sputum. When perforation occurs with the pleura full of oil, whether due to faulty technic or not, it is very dangerous because the oil may flood the opposite lung and cause death by suffocation. At the instant of recognition of this accident the oil must be removed. When caused by perforation of a subpleural focus due to evolution of the disease, the opening is usually large and gaping, and early thoracoplasty is advisable. Bronchopleural fistula may also be due to overpressure and to wounding of the lung by the needle. The latter is inexcusable. Küss advises using a needle whose length is just the thickness of the chest wall. At all times during the injection of oil one must be sure that the needle is in air or oil by aspirating a little before each syringeful of oil is injected. If the perforation is due to too high pressure, either of oil or of the trapped gas, it may not be large and the symptoms may be controlled by immediate aspiration. The following case illustrates the danger of overpressure.

Case 2.—H. S. began artificial pneumothorax on the left in November, 1926. Serous effusion developed in August, 1927, being aspirated in September. The general condition was good but, cavity not being completely collapsed, the adhesions were cauterized in February and in March, 1928. The latter was followed by a Staphylococcus aureus empyema. This infection was controlled by means of gentian violet by June but a needle abscess became a fistula through which the thin pleural fluid drained occasionally. Phrenic exairesis was performed in September, 1928. The cavity was closed, the lung well collapsed, and no refills were made after September. During the winter the general condition was excellent although the chest-wall fistula occasionally drained, the amount becoming less and less. During the active phase of the empyema a slight spread of disease had occurred in the right lung and this had improved. In April, 1929, the lung had not expanded at all, the pleura containing gas and a little fluid. Tubercle bacilli were present in it but no other organisms. There was a continual pulling sensation in the left chest. No evidence of bronchopleural fistula had ever been seen. Oleothorax was advised to avoid a possible thoracoplasty and to try to control the thin tuberculous pus. On April 20, 1929, the pleura-cutaneous fistula having been closed recently, the fluid was removed and 650 c.c. 5 per cent gomenol in paraffin oil were injected wth a one-needle technic.

After each injection of 50 c.c. of oil, gas was withdrawn. This filled the pleura to about the level of the second rib anteriorly. As the last syringeful was being injected the patient had a sharp, spasmodic cough and tasted gomenol but did not expectorate any oil. Oil was quickly withdrawn and the symptoms vanished. The gas pressure was left neutral. But he felt a slight fulness in the right anterior chest toward the base and more râles than usual were immediately heard here; they disappeared in a week and subsequent films showed no new shadows. Three days later he had a hard coughing attack and the gas pressure was found to be +9 to +4. Gas was withdrawn and this immediately stopped the cough and gurgling sounds. Again on April 26 he had another hard coughing spell and 250 c.c. of oil were then removed. The temperature rose to 100° and 101° until May 3 when the chest wall fistula opened from which were discharged oil and some thin pus. It then dropped to normal and the cough and expectoration again disappeared. Occasionally periods of slight discharge through the chest wall recurred. Taste of oil without visible globules or staining of the sputum cup occurred until November. Today the lung has partially expanded, gas and a little fluid are present and no discharge through the chest wall has occurred for over a year. The patient is clinically well.

In this case too much oil was injected at one time, too much, at least, with a one-needle technic. The evidence of perforation into the lung was seen when the oil reached the level of the cauterized adhesion. The empyema after cauterization and the persistence of gas in the pleura to date make one wonder whether a small perforation persisted after the cauterization or whether this left a weakened area. The attacks of spasmodic coughing during the days following injections were probably due to pleurisy with effusion pushing the oil level up to the perforation and compressing gas above it.

When oleothorax is used to try to control an established perforation one must be very cautious. It is always advisable here to use two needles. If the perforation is valvular or temporarily closed, it may be reopened by failing to control the overlying gas pressure. I had such an accident. After a momentary feeling of pressure, the patient coughed up a few drops of oil. Or, if the bronchopleural fistula is patent and large, one may incautiously force oil through the lung. It may be coughed up or it may enter the other lung where it may cause a bronchopneumonia (due to oil) or it may carry tubercle bacilli with pus from the pleura into the lung.

Oil embolism may occur. Oil may enter the circulation through the needle entering a vessel and, I believe, oil under pressure may be forced into newly formed capillaries.

CASE 3.—H. McC. developed pulmonary tuberculosis in 1919. Tuberculous spondylitis was diagnosed in 1922 and later healed. With chronic cavernous tuberculosis of both lungs, he did part-time work and had occasional small hemoptyses without sequela. In February, 1928, artificial pneumothorax was begun on the left to control these hemoptyses. With an 80 per cent compression the cavities were partially collapsed and bleeding was less in amount and in frequency. Serous effusion occurred in November, 1928. Several aspirations were performed. The fluid contained fibrin clots and by September, 1929, was purulent. Temperature was normal. In November pus filled the slowly diminishing pocket and nothing further was done until February, 1930, when it was decided to replace the pus with oil to maintain some compression of the left lung with the intention of trying later a partial pneumo-

thorax on the right to control the progressively enlarging cavities there. With difficulty 250 c.c. of thick pus were removed. Patient and operator both becoming tired, I foolishly determined before stopping to insert some oil. Not enough pus had been removed to get a gas pressure reading although air had been introduced. An adherent tongue of lung was in the vicinity of the needle. Although I had attempted to avoid this area some expansion may have occurred under the pus, for I believe that the needle point must have slipped into a vessel in the adhesion. After 150 c.c. of oil had been introduced with ease, the patient complained of feeling faint and became slightly pale but not cyanotic. He was immediately placed in a horizontal position. He appeared dazed but was not unconscious and responded to questions. No convulsion or muscle twitching occurred. He had no cough with the attack and no oil was ever tasted or seen in the sputum. Pulse was 80 per minute and strong; breathing natural. He then said that he felt numb in the back of the head. He could not move the right arm and leg although the reflexes were normal. When told to move the right arm he moved the left. Five minutes later he could move both right arm and leg. The right pupil was dilated slightly and vision was a little blurred. In another five minutes he felt all right, although the right arm felt slightly weak and he was tired. No further complication occurred. Fluoroscopic examinations showed a small amount of pus at the bottom of the pleura with oil above it. He resumed his part-time work later. Hemoptyses began again in November, 1930, and he died from a large one in July, 1931.

In the following case the oil caused a fatal pathologic lesion which I have not seen described elsewhere. This and several theoretical considerations about the indications and method of installation in this case deserve a detailed description.

CASE 4.—The patient, a young woman, developed dry pleurisy on the left and was admitted at Trudeau Sanatorium in May, 1929. Classified, moderately advanced, group A. Tubercle bacilli were never found in the sputum. Both lungs were infiltrated above the third rib without râles. She never regained full strength and the blood counts indicated slight activity of disease throughout the course. In December, 1929, and January, 1930, the sputum was occasionally blood-streaked. A small cavity developed in the left lung. She came under my care in January, 1930, after a recent slight hemoptysis. Although she was well nourished and had good color, very little mucosalivary sputum without tubercle bacilli and normal temperature and pulse rates, she had no strength. Small hemoptyses occurred in March but the cavity continued to contract and in May could not be seen. After mid-May she occasionally expectorated a small piece of thick sputum. Blood-streaked sputum in small amount occurred regularly with menses and occasionally at other times. Pleurisy on the left began on June 22 and later effusion was diagnosed with temperature 102°. An x-ray film then showed a reappearance of the small ring which was interpreted as a cavity. I decided then to keep the left lung compressed with artificial pneumothorax. On July 3, when the temperature was 102.4°, 1250 c.c. of serous fluid were removed and 1150 c.c. of oxygen instilled with a final pressure of +4 to −2. I feared quick reexpansion and adhesions on account of the pleurisy. On July 5 the readings were −2 to −4; 225 c.c. air; +12 to +10; on July 7 +2 to −4; 25 c.c. air; +12 to +8. On July 9 it was evident that the lung was reexpanding in spite of pressure. A thin film of fluid overlay the expanding lung. Punctures were made in various sites and small amounts of air given until July 18. The temperature fell to normal on July 19. A compressive oleothorax was decided upon in an attempt to reestablish the collapse, for with it the patient had lost her sputum, recently purulent. It would be necessary to have the oil under positive pressure in the hope that it might

force apart the newly formed adhesions and eventually close the cavity. Also, it seemed necessary to push oil refills more quickly than usual on account of the rapid reexpansion of the lung and the pocketing of the fluid. On July 22 with the patient sitting, the needle was inserted at the level of the sixth dorsal spine. This spot was chosen because at the last air refill a manometric reading had been obtained here and I wanted to be able to take pressure readings easily. One hundred and ten c.c. of 5 per cent gomenol in paraffin were injected by syringe, removing air alternately, and the final pressure was −2. That evening the temperature rose to 99.4° but dropped to normal on the next day. On the twenty-fourth, 125 c.c. of oil were introduced at the same level. When pressure oleothorax is used Küss advises testing the relative pressure by connecting to the needle, by means of a short piece of rubber tubing,

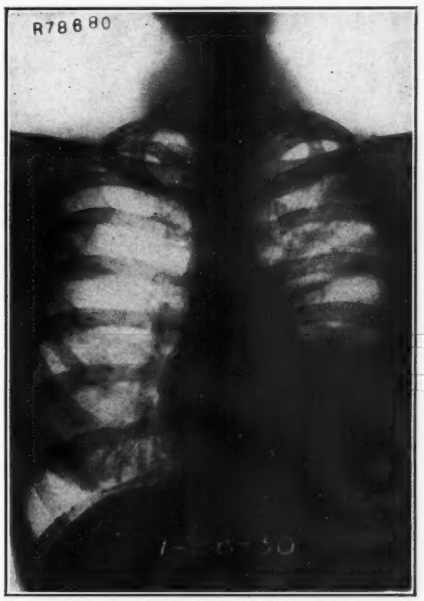


Fig. 1.—Right lung: moderate infiltration to third rib. Left lung: dense shadow below fourth rib probably thick pocket of oil as no effusion was found at autopsy. Thin shadow of oil to second rib, with gas pocket above (level oil line). Apex uncollapsed. Ring at second intercostal space interpreted as cavity but no compressed cavity seen at autopsy.

an upright glass tube of narrow calibre. When oil is under pressure it will rise into the tube slowly and, given time for leveling, one can measure its height and have a comparative value for subsequent injections. After 125 c.c. of oil had been introduced oil rose in the tube 19 cm. Five c.c. were removed and the end of the needle was then in the gas pocket; gas was removed and a neutral pressure obtained with a water manometer. On the twenty-sixth, introducing the needle at the same level, it entered the gas pocket. The oil seemed to have settled lower into the pleural pocket. After introducing 250 c.c. of oil, removing gas as long as the oil was below the needle, oil flowed up the Küss tube 11 cm. By percussing, it seemed to fill the pleura to the third dorsal spine. X-ray films were taken after each filling. At this time a dense shadow interpreted as effusion was seen at the base, oil surmounting it

to the second rib anteriorly. The ring at the second interspace, interpreted as cavity, had not been collapsed (Fig. 1). Some minute petechiae were observed on the arms, chest and thighs, but as the patient felt well no significance was attached to them. On the twenty-ninth the oil had again settled, for the needle entered gas at the same level. After injecting 500 c.c. of 2 per cent gomenolated oil the final pressure on the Küss tube was +26. Temperature was 99.6° to 100°. The patient was perfectly comfortable and had no cough or expectoration. On the thirty-first, introducing the needle at the same level with the patient in the sitting position as usual, the initial gas pressure was −2 to −6, indicating that the oil had settled again. After withdrawing gas until the pressure was −4 to −10, I then gave 260 c.c. of oil, when the oil rose in the tube 21 cm. At this point the patient felt a little faint but quickly recovered when placed prone. An x-ray film then showed a good filling with

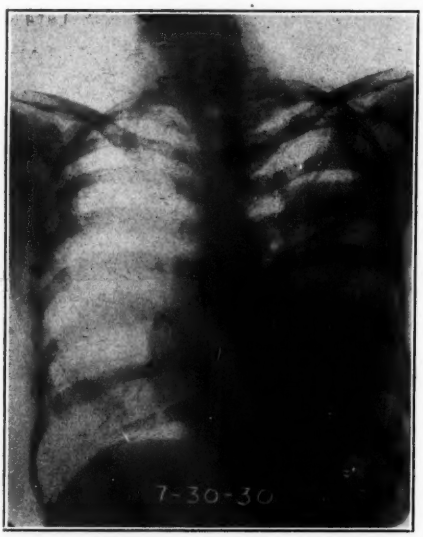


Fig. 2.—Better oil filling to second intercostal space. Air pocket and uncollapsed apex above. Ring gone.

disappearance of the cavity (Fig. 2). The right lung had not changed during this time. Menses, which were delayed two weeks, were established on August 1, at which time she spat up a clot of blood, a common occurrence with her with onset of menses. The temperature rose to 101°. As she was accustomed to feeling miserable at the menstrual period, she did not advise me of these symptoms. When I called on the second I was shocked to see her condition which had changed abruptly. Temperature was 102.4° and the respiratory rate rapid. She complained of nausea, vomiting, and extreme weakness. Many moderately coarse râles were heard throughout the right lung and new petechiae had erupted in the skin. An x-ray film showed marked clouding irregularly scattered throughout the right lung; the left showed the shadow of a small effusion and oil filled the remainder of the space (Fig. 3). No oil had been expectorated. The diagnosis was acute pneumonia, the change being considered too rapid for tuberculosis. That night she became cyanotic with marked dyspnea and died suddenly. No nervous symptoms were observed.

D. E. Cummings of the Saranac Lake Laboratory, performed the autopsy. I am obligated to him for his correct diagnosis and careful study and for the following notes. Many small hemorrhages had occurred into the skin and mesentery. The spleen, kidneys and liver were congested in areas. The stomach contained 500 c.c. of clotted blood and the mucosa here and in the duodenum and jejunum was markedly congested. The terminal ileum showed some tuberculous ulcers in all stages of activity. The right lung was consolidated and colored a deep purplish red. Around the healing tubercles at the apex the consolidation was less marked and the lung was slightly crepitant. Tracheobronchial lymph nodes were large, edematous, deep purplish red in color and without evidence of tuberculosis. The left pleural cavity consisted of many intercommunicating pockets formed by plaque-like adhesions. These pockets were full of clear oil, no pleural fluid being seen. The parietal pleura

Fig. 3.—Oil at higher level. Heart further to right. Irregular mottling throughout right lung.

was thickened and partially caseous. The left lung was well collapsed and atelectatic except at the apex where it was adherent to the chest wall. It was not as markedly consolidated as the right but was of the same color. The cavity could not be found after careful search. The tuberculosis in the upper lobe did not appear to be progressive (Fig. 4). Smears made from the cut surfaces of the lungs and lymph nodes and with heart's blood contained no acid-fast or Gram-stained organisms. Wright's stain of the heart's blood showed practically a normal white cell distribution. Frozen sections of tissue from the right lung and stomach were stained with Soudan III and fat-staining material was found in large amount in the capillaries.

Dr. L. U. Gardner's report on the sections of fixed tissues follows:

"*Lungs.*—There is extensive engorgement of both arteries and veins and very extensive hemorrhage into the air spaces from cartilaginous bronchi to alveoli. Evidence of injury to the blood vessels is lacking. No pneumonic exudate is dis-

closed. Stains for fat demonstrate heavy deposits in all layers of the caseous pleurisy. This occurs between the tissues. In other places, where the pleura is composed of vascular granulation tissue, there is fat within the dilated vessels. Deeper in the lung, fat is most abundant in the foci of hemorrhage where it is found both within blood vessels and within the alveoli. There is no indication that more fat occurs in the alveoli immediately beneath the pleura on the side of oleothorax than at other points in the lung.

"The tuberculous process consists of fibrous and fibrocaseous tubercles which are judged to be relatively inactive although the hemorrhage greatly obscures the picture. A striking feature is the presence of injected capillaries which run through

Fig. 4.—Pleural pocket obliterated above second rib level. Parietal pleura caseous, thickened and shaggy, held in place by glass rods. Fibrinofibrous septa cross the pleural space.

the tubercles up to the caseous zone. Of passing interest is the presence of a ring, fat staining, in the area of living epithelioid cells between caseation and the granulation tissue periphery of the tubercles."

This was a case of oil embolism without cerebral symptoms which caused death by diffuse hemorrhage into the pulmonary tissues. The gross appearance was that of red hepatization of large lobular areas. The petechiae of the skin, first seen four days after the first injection of oil and eight days before death, were probably due to oil emboli. At autopsy oil was found in capillaries in hemorrhagic and congested areas in various organs. It seems most probable that the oil, which was under pressure during the injection of July 24 (although the gas pressure was lowered at the end of the seance), must have entered the circulation through the newly formed ves-

sels of the soft pleural adhesions. The catastrophe occurred with the onset of menses at which time it was usual for some pulmonary congestion to occur, for she regularly spat blood-streaked sputum with menses. This increased congestion may have favored the oil entering thin-walled capillaries, for the pleura was undergoing fibrosis. This fatality should make one cautious about using "compressive" oleothorax in the face of a recent pleural effusion. The assumed indications for the very frequent refills (the quick expansion, the rapid failure of artificial pneumothorax, and the necessity of closing a cavity) may not have been logically grounded.

Fontaine says that phlegmon of the chest wall has occurred from olive oil of poor quality (rancid, improperly prepared). She has heard of only one case of paraffinoma and that in the mediastinum. Dumarest mentions paraffinoma of the chest wall. A minor inconvenience is that, with oil in the pleural cavity, compression of the lung cannot be watched as easily as with air. Pleura-cutaneous fistula occurred in 5 of 150 cases (Marie, Fontaine). This is usually due to tuberculous infection of the needle tract. To avoid this accident one may use the usual methods: cleansing interior of needle before withdrawal, washing pleura free of pus, injection of oil high in axilla, using a small needle. Oil may be coughed out and lie under the skin. To avoid this Fontaine gives the following directions:

1. Never make blockage with positive pressure in patients too emaciated or subject to attacks of repeated cough.
2. Always give a sedative for cough on the days following injection to avoid increased pressure.
3. Observe with care the secondary fluid reactions and aspirate fluid if necessary.
4. Use a compressive bandage after massage of the tract.
5. Advice absolute repose on the side opposite oleothorax for a minimum of two hours.

## CONCLUSIONS

Oleothorax, while a valuable adjunct to pulmonary collapse therapy, may be dangerous and requires a very careful technic. It causes minor inconveniences and accidents: paraffinoma, pleura-cutaneous fistula, febrile and exudative pleural reactions, difficult observation of the lung.

Graver accidents (bronchopleural perforation, oil embolism) may be fatal.

Several illustrative cases are recorded.

## REFERENCES

Important references on technic, complications and results:
Küss: Arch. med.-chir. de l'app. resp. **1**: 203, 1926. Rev. de la Tuberc. **7**: 100 and 649, 1926. Ibid. **9**: 714, 1928.
Bernou: Ibid. **7**: 345, 1926. Ibid. **9**: 742, 1928.
Bernou and Laurens: J. des Praticiens, 1923, p. 196.
Fontaine: Oleothorax, Gaston Dion and Co., Paris, 1929.
Marie: Thèse de Paris, 1929.
Labesse and Marie: Rev. de la Tuberc. **11**: 648, 1930.

# EXPERIMENTAL FIXATION OF THE MEDIASTINUM

MAURICE BERCK, M.D.,* ST. LOUIS

THE significance of mobility of the mediastinum in the alterations resulting from variations in intrapleural pressure led to investigations designed to attempt fixation of the mediastinal structures. That such fixation of the mediastinum would be of service is clear when consideration is taken of the familiar rôle played by the mobile mediastinum in the pathologic alterations of the physiologic mechanisms involved in open pneumothorax and collapse therapy. The importance of the fixated mediastinum with the theoretical considerations underlying the phenomena noted with the mobile mediastinum have been elaborated in several papers by Graham upon which is based the modern treatment of empyema. Graham has shown that in the normal thorax the mediastinum offers so little resistance to changes of intrapleural pressure that any slight or moderate change of pressure in one pleural cavity is directly translated into nearly equivalent pressure effects in the other. However, if the mediastinum is fixed, stabilized—rendered immobile—none of the ill effects of open pneumothorax will be noted.

The deleterious effects of excess mobility of the mediastinum—the "flapping" or "flutter" of the mediastinum—are principally noted in open pneumothorax in which the great and rapid encroachment upon vital capacity results in great respiratory distress and even asphyxial death; and in decostalizing operations (thoracoplasty) when the "deribbed" cage, deprived of its bony support, swings in and out freely with the respiratory movements, being drawn in with inspiration and forced out with expiration passively, due to the respiratory efforts of the opposite intact chest wall with its action transmitted directly through a lax mediastinum. This paradoxical respiration is possible only because of the nonfixated mediastinum and has as its accompaniment and sequelae, rapid reduction in vital capacity through encroachment upon the opposite side which may go on to respiratory failure, circulatory embarrassment and perhaps circulatory failure by the effects upon the heart, great vessels and lesser circulation through the rapid forceful changes in position of the mediastinal septum. Great impairment of the coughing and expectorating mechanisms may occur with propulsion into and retention of bronchial secretions in hitherto unaffected portions of the lung with not infrequent aspiration and pneumonia. The tuberculous lung does not derive the rest for which the operative attack was designed

*Chest Service, Department of Surgery, Washington University Medical School, and Barnes Hospital, St. Louis.

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and the contralateral lung must overwork more than is safe for the few tuberculous lesions that it contains.

The importance of the fixed mediastinum and measures to control its excursions in thoracic operations can hardly be overemphasized. In nearly every type of major thoracic operation the factor of the mobile mediastinum comes into play. In pneumothorax therapy the danger is ever present of high tension positive intrapleural pressure, artificial, or through complicating spontaneous pneumothorax with resultant dislocation of the mediastinal septum and its well known train of symptoms. In thoracoplasty, as has been pointed out repeatedly by Archibald and more recently abroad in reviews of large series of thoracoplasties by Hauke, Jessen and Nissen, the necessity of a fixed mediastinum with tracheal retraction to the diseased side is of paramount and primary importance. In a large series of thoracoplasties recently reported Jessen stated that cardiovascular accidents were the chief cause of immediate postoperative mortality. He stressed mediastinal fixation as an essential requisite for collapse therapy because of the pronounced deleterious effects of mediastinal flutter upon the heart and great vessels and hence upon the circulation. Reports upon plombage from Sauerbruch's clinic stress the importance of mediastinal fixation to avert mediastinal shift with its consequent cardiac embarrassment and tracheal stenosis. Even more pertinently, it is pointed out that in the absence of a fixed mediastinum effacement of a pulmonary cavity is not achieved but instead the pressure of the plombe merely displaces the lung containing the cavity beneath the plombe mesially, shifting the mediastinum over and results in transposition of the cavity, not in its obliteration.

Mobility of the mediastinum depends upon the flexibility of its tissues and the lack of pleural or pericardiopleural adhesions. The degree to which operative procedures may alter the position of the mediastinum is referable primarily to changes in intrapleural pressures within the two pleural sacs and their relation to the firmness with which the mediastinum is held in place by adhesions or inflammatory changes within itself and the extent to which the mediastinum has been pulled to one side by shrinking scar tissue in the lung and pleura. The entire septum may be shifted or only a part of it.

The upper anterior portion of the anterior mediastinum between the first and third ribs (the site of the atrophied thymus) represents the weakest point in the mediastinal barrier; here the right and left mediastinal pleural laminae come in closest contact and are separated by only a small amount of areolar tissue. The retrocardiac area between the fifth and tenth thoracic vertebrae is another "weak spot." However, nearly all of the mediastinal herniations due to pneumothorax have been situated in the upper anterior "weak spot" of the anterior mediastinum.

Adequate fixation of the mediastinum was therefore thought to be pos-

sible if a fixating infiltration of the "weak" portion of the mediastinal barrier—the anterior mediastinum—could be secured.

## ANATOMY

The anterior mediastinum is bounded anteriorly by the sternum. The space is limited anteriorly by the muscles and periosteum lining the posterior surface of the sternum. It is narrowest both in the sagittal and transverse diameters behind the upper extremity of the gladiolus since the two mediastinal laminae are in contact in this situation immediately behind the bone, but superiorly and especially inferiorly, it is broader as the two mediastinal laminae diverge so that the pericardium is uncovered by the pleura behind the lower portion of the manubrium and the fifth and sixth costal cartilages. The mediastinal septum is consequently slightly obliquely placed and its lower portion extends for a considerable distance to the left. The anterior mediastinum attains a considerable sagittal diameter only behind the manubrium, the remaining cavity being filled with loose areolar tissue. The upper portion contains the thymus gland or the fatty remnants of it. A few lymph nodes of the sternal, internal mammary and anterior mediastinal groups are present.

The internal mammary vessels run nearly parallel to the lateral margin of the sternum, separated from it laterally by 1 to 2 cm. The right mediastinal pleura almost exactly follows the right anterior pulmonary border and rarely passes the midline of the sternum, but the left mediastinal pleura usually extends only to the left sternal margin from which it deviates laterally in the region of the fifth or sixth costal cartilage, so that the pericardium is uncovered by pleura in this situation. In the anterior portion of the superior mediastinum the two mediastinal laminae diverge following the convexity of the apices. The two pleural laminae may be nearly in contact between the second and fourth spaces (Fig. 1).

The anterior mediastinum may vary considerably because of variations in the line of the pleural reflections. It is to be especially noted that the communication on each side with the space external to the parietal pleura is particularly free. The parietal pleura has no firm connection with the sternum but is rather widely separated from it by loose areolar tissue, the parietal pleura in fact becoming adherent only to the line of the costal cartilages.

In experimental fixation of the mediastinum the following features were considered:

1. The method and substances producing fixation must be innocuous, particularly in the sense that the tissue reaction to them must not produce Pick's syndrome of adhesive mediastinopericarditis. Furthermore, a prerequisite is that the fixating agent used be bland, nonirritating and nonprovocative of an acute inflammatory reaction.

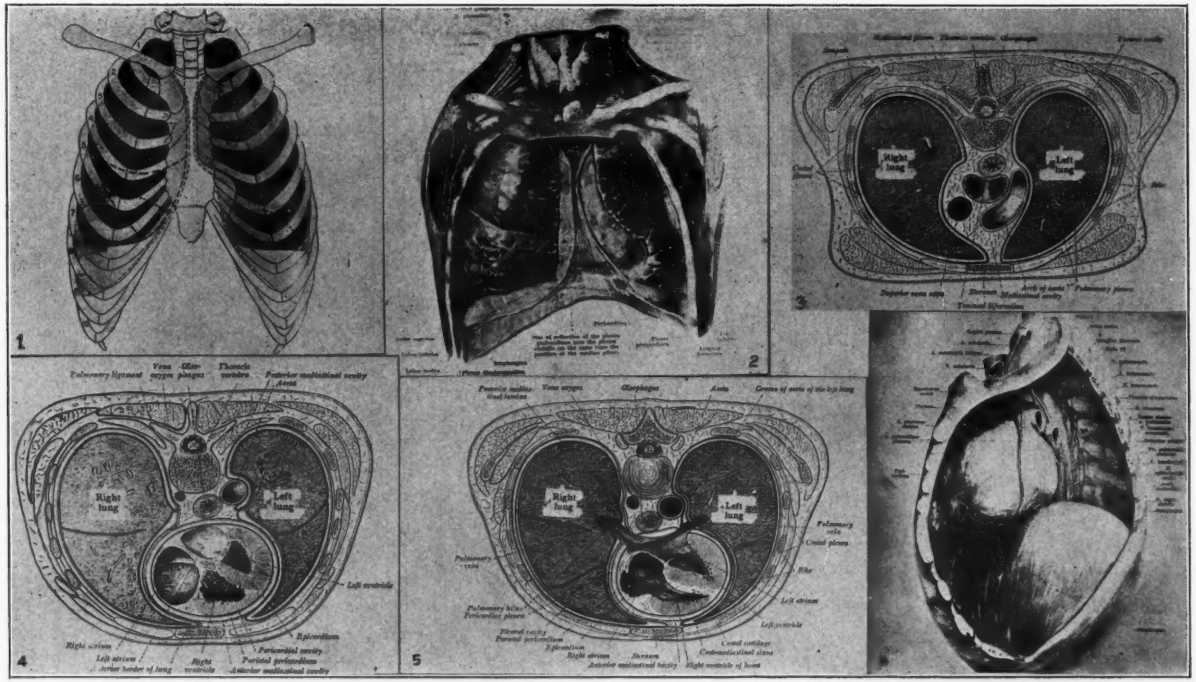


Fig. 1.—Illustrations to demonstrate the anatomy of anterior mediastinal space and anterior portion of superior mediastinal space.

*1.* Diagram to represent relations of pleural reflections to the thoracic cage (Sobotta).

*2.* Anterior mediastinum revealed after anterior thoracic cage has been removed showing apposition of the two pleural laminae between II and III intercostal spaces and the widening of the anterior mediastinal space superiorly and inferiorly ("undefended" precardiac space) (Spalteholtz).

*3.* Cross-section of thorax at level of inferior margin of manubrium (Sobotta).

*4.* Cross-section of thorax at level of "undefended" precardiac space (Sobotta).

*5.* Cross-section of thorax at level of apposition of pleural laminae (Sobotta).

*6.* Sagittal representation of thoracic viscera demonstrating position and area occupied by anterior portion of superior mediastinum and anterior mediastinum (Spalteholtz).

2. The duration of fixation should be of variable degree inasmuch as the fixation required for a surgical open pneumothorax (thoracotomy) to avert mediastinal flutter should be complete but short-lived, whereas

Fig. 2.—Photographs of fixating infiltrations (rabbit) of anterior mediastinum and anterior portion of superior mediastinum.
*1.* Paraffin oil (15 c.c.) injected 2 months previously.
*2.* Agar (15 c.c.) injected 2 months previously.
*3.* Agar (15 c.c.) injected 2½ months previously.
*4.* Paraffin oil (15 c.c.) injected 3 months previously.

the fixation necessary for an extrapleural thoracoplasty should be permanent as well as initially complete.

3. The fixating agent if short-lived and quickly absorbed, should be of high viscosity so as to act immediately as an effective "mediastinal

splint," whereas other fixating agents for permanent residence should possess in addition to viscosity for initial fixation an inert nature and be but slowly provocative of an innocuous, chronic productive inflammatory reaction.

## EXPERIMENTS (ON THE RABBIT)

The following experiments to determine the feasibility of such a method of fixation were undertaken. Adult rabbits were used because of the relatively strong development of the septal structures. Dogs were not used because of the tenuous, filamentous nature of their mediastina rendering them incapable of infiltration.

Dissections in the rabbit revealed the anterior mediastinal space to be potentially large with ready extension to the anterior portion of the superior mediastinum. This space accommodates 15 c.c. of fluid if slowly injected. Roentgenograms after injection of this space with radio-opaque fluids were made which demonstrated the feasibility of filling this space with substances which either by action as mechanical barriers or by provocation of an innocuous slowly progressive productive inflammation would secure fixation of the mediastinum.

The standard of control was the shift of the mediastinum (adult rabbits) in response to 20 c.c. of air introduced through the left seventh intercostal space. Uniformly in the six control animals such a pneumothorax succeeded in shifting the mediastinum far to the right so that the left cardiac border coincided with the left margin of the vertebral column. (Fig. 3, *A, B, C.*)

The following injections of the anterior space and the anterior portion of the superior space were then made through a needle introduced on both sides of the xyphoid process.

(1) 15 c.c. 30% acacia solution (4 rabbits)
(2) 15 c.c. Agar (6 rabbits) (melted to body temperature)
(3) 15 c.c. Paraffin oil (6 rabbits)
(4) 15 c.c. Suspension of silicon dioxide in paraffin oil (5 grams in 15 c.c.) (3 rabbits)
(5) 15 c.c. Suspension of silicon dioxide in water (5 grams in 15 c.c.) (2 rabbits)
(6) 15 c.c. Suspension of sodium and potassium silicate in paraffin oil (5 grams in 15 c.c.) (3 rabbits)
(7) 15 c.c. Suspension of sodium and potassium silicate in water (5 grams in 15 c.c.) (3 rabbits)

Roentgenograms were then made of the position of the fixated mediastina which were observed to be widened moderately and in the midline. After variable periods of time (six hours to six months) dependent on the type of fixatant used, left pneumothoraces were produced in the identical manner as in the control unfixated rabbits, (20 c.c. left seventh intercostal space). Roentgenograms were then immediately made. (Fig. 3 shows comparison of effect of pneumothorax on normal and unfixated mediastina.)

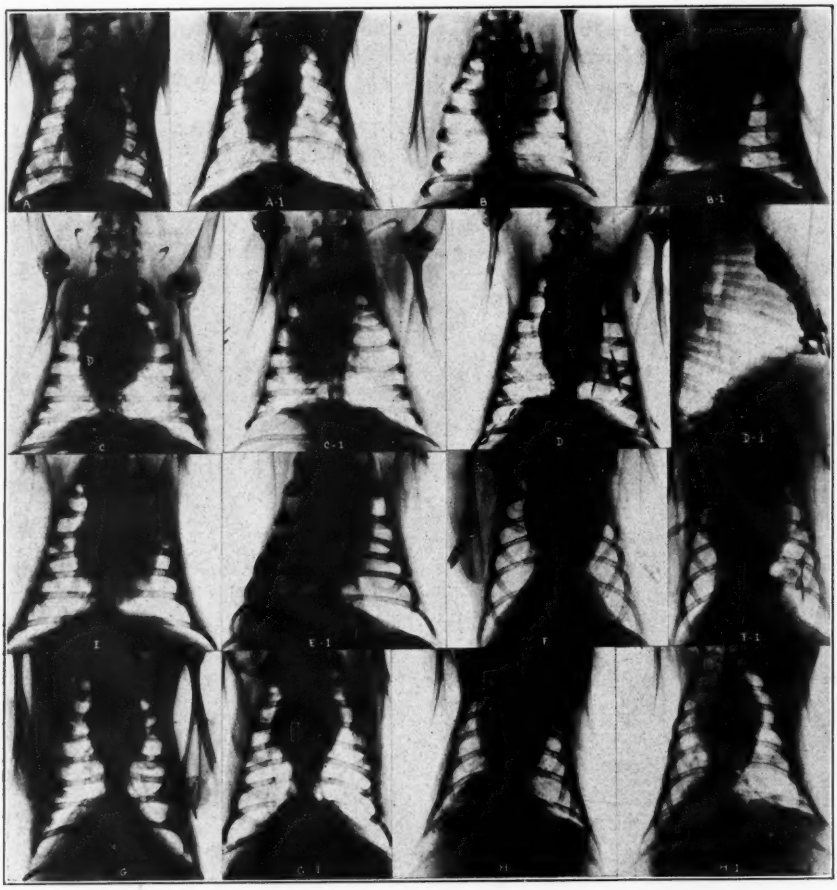

Fig. 3.—Artificial mediastinal fixation of the rabbit.

The first three sets of roentgenograms are controls. They illustrate effects of pneumothorax (20 c.c.) in shifting the mediastinum of the normal rabbit. The last four sets demonstrate effects of same amount of pneumothorax when artificial fixation of the mediastinum has been brought about.

*A.* Normal rabbit. Note central position of mediastinal septum and its contained structures.

*A-1.* Induction of left-sided pneumothorax (20 c.c.) in the normal rabbit. Note marked shift of the mediastinum to the right. (Control.)

*B.* Normal rabbit.

*B-1.* Induction of left-sided pneumothorax (20 c.c.) in the normal rabbit. Note the extreme shift of the mediastinal septum to the right. (Control.)

*C.* Normal rabbit.

*C-1.* Induction of left-sided pneumothorax (20 c.c.) in the normal rabbit. Note marked shift of the mediastinal septum to the right. (Control.)

*D.* Artificial fixation of the anterior mediastinum and anterior portion of the superior mediastinum of the rabbit with lipiodol (15 c.c.) (for radio-opacity). Anteroposterior view. Note distensibility and potential size of anterior, mediastinal space. (The linear shadows at the left base are the wires of number tags in the ears of the rabbit.)

*D-1.* Lateral view of *D* showing the anatomic position occupied by the fixating infiltration of the anterior mediastinum.

*E.* Artificial fixation of mediastinum of rabbit with 15 c.c. of 30 per cent acacia.

*E-1.* Induction of left-sided pneumothorax (20 c.c.) in rabbit with artificial fixation of mediastinum (15 c.c. of 30 per cent acacia). Compare position of fixated mediastinum with control normal unfixated mediastina, Figs. *A-1, B-1, C-1.*

Autopsies performed on a number of the animals have shown the fixating material to be restricted to the distended anterior space and anterior portion of the superior space. The acacia disappears entirely after thirty-six hours; the agar has remained for at least 2½ months; the paraffin oil persists for several months and engenders a low grade fibrotic reaction; the silicon dioxide and silicate suspensions in oil provoke a more pronounced fibrotic reaction; the suspensions of silicon dioxide and the silicates in water arouse a more intense productive, and in some cases exudative mediastinitis (Fig. 2).

The results as illustrated by the roentgenograms (Fig. 3) may be summarized briefly as follows:

1. Fixation of the mediastinum of the rabbit in the midline sufficient to resist the pressure of a 20 c.c. left pneumothorax, which in unfixated rabbits causes shift of the mediastinum far to the right, can be secured by injections of the anterior mediastinal space and anterior portion of the superior space with 15 c.c. of acacia (30 per cent), agar, paraffin oil, suspensions of silicon dioxide and silicates in paraffin oil.

2. Complete fixations of variable duration can be secured. The fixating effect of acacia is evanescent whereas that of agar is longer and that of paraffin oil seems permanent. The admixture of silicon dioxide and silicates to the paraffin oil slowly arouses a productive inflammatory response.

Upon the basis of the results of the animal experimentation investigation was undertaken to attempt fixation of the mediastinum of the fresh human cadaver. Thus far six cadavers (none later than two hours postmortem) have been studied.

## EXPERIMENTS (ON THE HUMAN CADAVER)

Dissection revealed the anterior mediastinum to be readily approachable by needle through two sites primarily; through the left sixth intercostal space and over the superior margin of the manubrium. Care must be taken that the needle is directed so as to hug the sternum closely and slip directly under it. (For this purpose a curved needle No. 16, angled at about 30° from the shaft, has been designed.) In the sixth space hugging the sternum closely the needle is superficial to the "undefended" portion of the pericardium, the pleura does not come into consideration being deflected to the left and the internal mammary vessels

*F.* Artificial fixation of mediastinum of rabbit with 15 c.c. of suspension of silicon dioxide in paraffin oil.

*F-1.* Induction of left-sided pneumothorax (20 c.c.) in rabbit with artificial fixation of mediastinum (15 c.c. of suspension of silicon dioxide in paraffin oil). Compare position of fixated mediastinum with control normal unfixated mediastina, Figs. *A-1, B-1, C-1.*

*G.* Artificial fixation of mediastinum with 15 c.c. of agar.

*G-1.* Induction of left-sided pneumothorax (20 c.c.) in rabbit with artificial fixation of mediastinum (15 c.c. of agar). Compare position of mediastinum with control normal unfixated mediastina, Figs. *A-1, B-1, C-1.*

*H.* Artificial fixation of mediastinum of rabbit with 15 c.c. of paraffin oil.

*H-1.* Induction of left-sided pneumothorax (20 c.c.) in rabbit with artificial fixation of mediastinum (15 c.c. of paraffin oil). Compare position of fixated mediastinum with control normal unfixated mediastina, Figs. *A-1, B-1, C-1.*

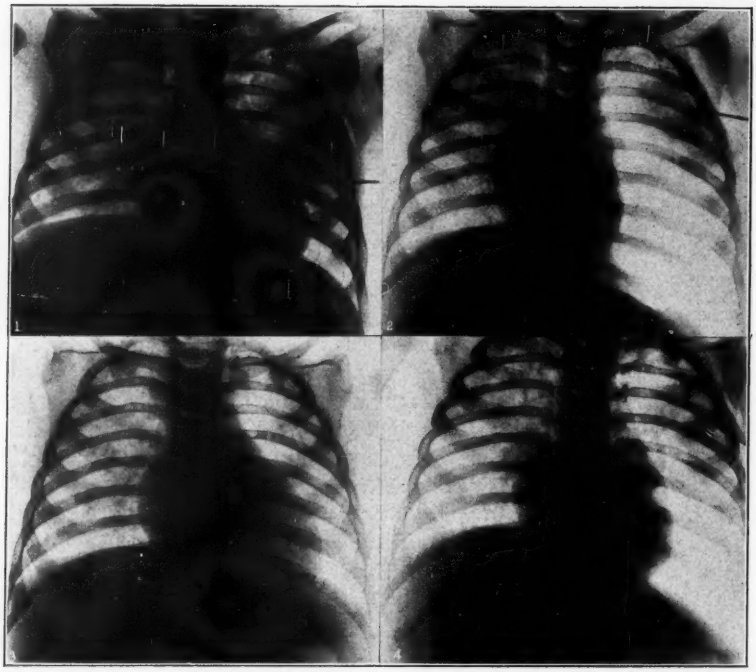

Fig. 4.—Artificial fixation of the mediastinum of the cadaver. Subject (M. F.) child three years old, death due to meningitis. Fixation and study performed twenty minutes after death.

*1.* Roentgenogram of chest (20 minutes postmortem).

*2.* Induction of left-sided pneumothorax (300 c.c.) showing marked mediastinal shift to the right.

*3.* Aspiration of pneumothorax (300 c.c.) and artificial fixation of mediastinum by anterior mediastinal infiltration with 80 c.c. of 30 per cent acacia.

*4.* Induction of left-sided pneumothorax (300 c.c.) after artificial fixation of mediastinum, showing relative immobility and fixation in the midline of mediastinal septum. Compare Figs. *2* and *4*.

are 1 to 2 cm. lateral to the margin of the sternum and thus easily avoided. When injecting over the jugular notch, if one directs the needle to follow the deep surface of the manubrium closely, no trouble need be expected from injury to the superior mediastinal structures. By this route of injection infiltrations of the anterior mediastinum and anterior portion of the superior mediastinum have been secured varying from 75 c.c. for infants to 250 c.c. for adults. As the fluid is injected it slowly dissects out the ramifications of the space and keeping behind it the needle may thus be slowly advanced until the entire space has been filled without injury to any structure. This has been confirmed in the dissecting room and by fluoroscopy with radio-opaque fluid.

The same experimental technic as was employed in the experiments upon the rabbits was utilized. A high tension pneumothorax (left-sided, fifth intercostal space, axilla) was induced sufficient to shift the medias-

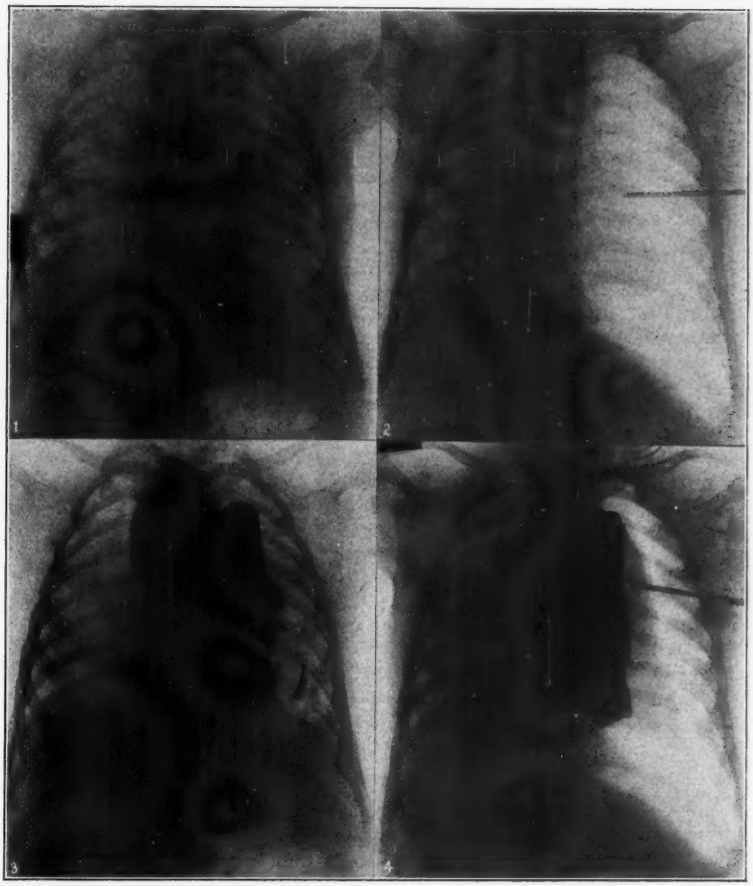

Fig. 5.—Artificial fixation of the mediastinum of the cadaver. Subject (S. G.), age four and one-half years, death due to meningitis. Fixation and study performed thirty minutes after death.

*1.* Roentgenogram of chest (30 minutes postmortem).

*2.* Induction of left-sided pneumothorax (320 c.c.) showing marked mediastinal shift to right and large pneumothorax pocket.

*3.* Aspiration of pneumothorax (320 c.c.) and artificial fixation of anterior mediastinum and anterior portion of superior mediastinum with radio-opaque mixture of paraffin oil and lipiodol (75 c.c.).

*4.* Induction of left-sided pneumothorax (320 c.c.) showing relative immobility and fixation in the midline of mediastinal septum (compare Figs. *2, 3, 4*).

tinal septum appreciably (fluoroscopy) (300 c.c. to 2000 c.c., varying with the subject). A roentgenogram was then taken. The pneumothorax was then aspirated completely, the amount of air aspirated being measured accurately. Infiltration of the anterior mediastinum and the anterior portion of the superior mediastinum through the sixth left intercostal space parasternally and over the jugular notch as above outlined

was then performed, using acacia (30 per cent) or paraffin oil, the injections varying with the size of the cadaver. Reinflations of air were then done in exactly the same manner as before, using the same amount as aspirated from the previous pneumothorax, and roentgenograms were immediately taken.

Uniformly a great degree of stability had been secured (Figs. 4 and 5). The locations of the fixatant infiltrate in the anterior mediastinum and anterior portion of the superior mediastinum were all checked by autopsy.

## CASE REPORT

To ascertain the clinical feasibility upon the living subject, very recently a patient with tuberculous empyema (T. S. Case 32565 Barnes Hospital) was subjected to mediastinal infiltration with acacia (30 per cent). Inasmuch as the animal experiments had shown rapid absorption of acacia in the mediastinum no permanent ill effects were anticipated. For this infiltration local anesthesia was employed (novocaine ½ per cent for skin and chest wall). No pain was experienced. Other than a pulse rise of 20 persisting for six hours no other symptoms were noted. No ill effects whatever occurred and the patient on subsequent oleothorax treatment for the empyema has improved appreciably in general condition. Roentgenograms showed slight widening of the mediastinum. This patient had widespread chronic pulmonary tuberculosis and tuberculous empyema of long standing. The mediastinum was presumably stabilized by chronic inflammation. No attempt was made to determine whether the mediastinal injection accomplished fixation inasmuch as the patient's conditions contraindicated collapse therapy. The injection was performed to demonstrate the clinical innocuousness of the procedure.

I wish to acknowledge with many thanks the hearty cooperation of the Department of Radiology, Dr. Sherwood Moore, Director, and the valuable assistance of Dr. Hugh Wilson of the Department of Radiology in securing the radiographs.

## SUMMARY

1. The rôle of the mobile mediastinum in thoracic surgery is discussed.

2. The anterior mediastinum is the weakest point of the mediastinal barrier and is easily approachable for fixating infiltration.

3. Infiltration of the anterior mediastinum and anterior portion of the superior mediastinum of rabbits and man with highly viscous fluids such as acacia, agar and paraffin oil is feasible and that of acacia seems noninjurious to man. A method for the infiltration is described.

4. In rabbits and in human cadavers, infiltration with acacia (30 per cent), agar and paraffin oil of the anterior mediastinum and anterior portion of the superior mediastinum suffices to secure sufficient fixation of the mediastinum to resist high pneumothorax pressures. With such fixation, innocuous in experiments, the mediastinal shift consequent to high tension pneumothorax did not occur.

## DISCUSSION

DR. EVARTS A. GRAHAM (St. Louis).—I simply want to say that I have watched the work of Dr. Berck all the way through. It has been done very carefully and cautiously, and I think the results which he has obtained are sufficiently striking to make the procedure of very distinct value.

The question was asked of me a moment ago, why Dr. Berck selected this particular human subject for the injection, that is, one in which apparently there was no necessity for fixing the mediastinum.

The reason for that was simply that it was necessary, in making the first injection, when there was some doubt perhaps as to whether or not the material might be harmful, to select a patient who was willing to have the injection made. That is why this particular patient was injected.

There is, of course, one possible danger to this procedure which comes to mind. That is, in those cases in which the mediastinum is abnormally developed, in which the pleural surfaces meet in the midline, as they occasionally do, there might be some danger of inadvertently injecting the pleural cavity with one of these solutions. However, even if that should happen, I do not believe it would be of serious consequence, because it would merely produce a pleural effusion. That would be perhaps the worst it would do. Perhaps pleural adhesions in some cases where this would be used would be desirable rather than objectionable.

DR. BERCK.—With regard to accidental entry into the pleural cavities with any of these fixatant solutions, I might say that a small amount of acacia, agar or paraffin oil would probably be noninjurious. Early in the experimental work on this subject, I was attracted by the fascinating chemical biology of pulmonary silicosis and employed suspensions of silica and silicates in the anterior mediastinum. However, the inflammatory reaction is too rapid and intense, I think, to be clinically feasible. I like to think of the action of mediastinal fixation as being that of a mediastinal "splint"—the fixatant fluid initially fills up the loose alveolar spaces of the anterior mediastinum and the anterior portion of the superior mediastinum, the "filling" perhaps exerting pressure on the heart anteroposteriorly obliterating the retrocardiac "weak spot"—and then later the fixatant fluid slowly engenders a low grade, slowly progressive, chronic productive inflammatory response for permanent mediastinal fixation. The acacia in contrast to agar and paraffin oil is of but short lived duration and is rather quickly absorbed.

# LEFT CONGENITAL DIAPHRAGMATIC HERNIA, IN A BABY OF THIRTEEN DAYS, OPERATED ON SUCCESSFULLY*

POL N. CORYLLOS, M.D., AND ABRAHAM TOW, M.D.
NEW YORK, N. Y.

CONGENITAL diaphragmatic hernia is a rather rare entity. The diagnosis, however, is not particularly difficult if the condition is only considered. As the lesion is usually left-sided, the existence of a dextrocardia, accompanied by bulging of the chest wall, dyspnea, cough, vomiting, and especially cyanosis, occurring in attacks, and promptly relieved with changing the position of the baby, and furthermore the physical signs, i. e., zones of dullness of breath sounds or tympany and presence of peculiar musical sounds on the affected side of the chest varying from one moment to the other, should suggest the possibility of a diaphragmatic hernia. Radiographic examination allows the final diagnosis; it shows the presence in the chest of fluid and gas, disposed in several fluid gas levels, and ingestion of an opaque meal reveals the presence of the intestine in the chest cavity.

When the intestine is herniated into the pleural cavity and especially when symptoms of partial obstruction are present, the only successful form of treatment is surgical intervention. Age should not be deterrent to surgical interference, if it is desired to improve the actual statistics, which show that 72 per cent of these cases are autopsy findings.

The following report is that of an infant who was successfully operated upon at the age of thirteen days. This is, we believe, the youngest case ever reported.

## CASE REPORT

J. S. was delivered on August 14, 1931, at Polyclinic Hospital, by Dr. E. H. Hawks. Birth weight was 6 pounds and 4 ounces. It was a full-term, low forceps delivery, and she was the first child. Physical examination was negative, but it was later noticed that she seemed to tire quickly when nursing. On the morning of the seventh day she had an attack of marked cyanosis. Examination showed a fairly well-developed and nourished female infant with moderate dyspnea. A diagnosis of cerebral hemorrhage was made, and 20 c.c. of mother's blood were given intramuscularly. The baby was put in Fowler's position and left absolutely alone for twenty-four hours. She had no attacks of cyanosis in that period, but dyspnea continued and she vomited occasionally. Thymus hypertrophy and cardiac lesion having been ruled out and having showed some dextrocardia present we suspected diaphragmatic hernia, and a radiogram of the chest was taken.

The film showed presence of gas and fluid in the left pleural cavity. A barium meal was given, and the diagnosis of diaphragmatic hernia with intestine present in the left thoracic cavity was made (Fig. 1). The attacks of cyanosis gradually grew

*Case presented before the New York Society for Thoracic Surgery, November 12, 1931.

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more frequent. The chest wall daily bulged more anteriorly, especially on the left side. The heart was pushed completely over to the right. Percussion of the left chest gradually became more tympanitic, and no breath sounds could be heard.

Surgical intervention was deemed imperative, and the baby was operated upon by one of us (P. N. C.); the weight was 5 pounds, 15 ounces.

*Operation.*—August 17, 1931. Oxygen-ether anesthesia was given by intratracheal insufflation.

Fig. 1.

Fig. 2.

Fig. 1.—Six hours after the ingestion of barium. The whole small intestine, the cecum, and the transverse colon are in the left chest.
Fig. 2.—Two months after operation. Left lung clear, no fluid present, heart in normal position. Baby in perfect condition.

Thoracotomy was done by incision on the eighth space from the costal angle to the anterior part of the space. No rib resection was performed. Tuffier's retractor was introduced into the opening and the intestine eviscerated from the thoracic cavity.

The whole of the small intestine, the cecum, the transverse and part of the descending colon and the spleen were in the chest. The opening of the diaphragm, situated in the posterolateral portion, was triangular in shape, with its base on the posterolateral wall of the chest. No adhesions were present between the intestine and the structures of the chest. The left lung was completely atelectatic. Reduction of the intestine through the diaphragmatic opening was found impossible, because of the position of the spleen, which was riding on the anterior edge of the opening and was solidly immobilized in that position by the pull exerted on it through the vasa breviora, by the greatly dilated stomach which remained in the abdomen; on the other hand the spleen was pressing upon and pushed by the intestine present in the diaphragmatic ring. After five minutes of gentle but vain efforts for reduction of

the spleen, the thoracic incision was prolonged upon the abdominal wall, left of the midline. The cartilage of the costal border was partially incised. With the index finger of the left hand introduced into the abdomen and the thumb introduced into the chest, the diaphragm being between the two fingers, the intestine was pushed backward, the diaphragmatic opening gently dilated and the spleen reduced into the abdominal cavity. By pushing the intestine through the thoracic cavity and pulling through the abdominal wound, the reduction was performed and the intestine kept in hot pads outside of the abdominal cavity. This was a long and laborious procedure during which the chest was widely open and the baby was breathing with only the right lung, the latter strongly handicapped because of the displacement of the mediastinum to the right side. From time to time the heart would slow down, and we would stop the reduction, covering the thoracic and abdominal openings with hot wet pads and then proceeding again. The opening of the diaphragm was sutured with running chromic catgut, the edges slightly overlapping, and reenforced with three stitches of dermal gut. The chest was closed without drainage by three dermal gut sutures passing around the seventh and eighth ribs; a continuous running suture of chromic catgut was used for the muscular layer. The reduction of the intestine into the abdomen was very difficult because of its distention and especially because of the extreme inflation of the stomach. A puncture with a fine needle in the stomach was made and the air aspirated, reducing the size of that organ and facilitating the reduction of the intestine and of the closure of the abdomen. Interrupted chromic catgut sutures taking together the peritoneum and muscles were used. The skin was closed with Michel clips and with a few silkworm gut retention stitches. No drainage.

At the end of the operation the baby was only moderately shocked, but it was cyanotic and breathing very rapidly. A Wassermann needle connected to the suction apparatus was introduced into the left chest through the eighth interspace and the air present in the left pleural cavity aspirated, in that way forcing the lung to expand. Immediately after this procedure respiration of the infant became easier and less rapid, and cyanosis disappeared almost completely. It was remarkable how well the little patient stood the ordeal of the operation, which lasted forty-five minutes.

*Postoperative Treatment.*—The usual supportive treatment of clyses was given postoperatively—oxygen and carbon dioxide, for two minutes every half hour, were administered for two days. Marked abdominal distention persisted for several days, but this finally subsided. X-ray picture six hours after the operation showed no air visible in the left chest, the left lung expanded and the heart was less displaced to the right side than before the operation. Two days later the heart was almost in normal position. A slight infection of the abdominal incision developed, and the left chest slowly filled up with fluid, and the heart was pushed upward and to the right. However, the general condition gradually improved, and two chest taps relieved the dyspnea due to the fluid. The chest wound also became slightly infected and this subsided slowly. Breast milk was used at first but a simple milk mixture replaced this. The patient was discharged six weeks after the operation, weighing 7 pounds and 1 ounce, and in good condition. There was still a slight discharge from the abdominal incision, but the left chest was free from fluid and the heart was in normal position.

Baby was seen again October 31; it was in perfect condition and the wound was completely healed. Fluoroscopic examination showed a normal chest, left diaphragm mobile, left lung well aerated, heart in normal position.

Although an operation of such magnitude seems at first sight incompatible with the resistance of a newborn baby, so that often internists

and surgeons hesitate to advise and perform it, we think that, when the condition is more closely analyzed and the seriousness of the prognosis considered, the operative dangers will appear less insuperable. In fact, it is obvious that the lung of the affected side being already collapsed, the negative intrapleural pressure being replaced by positive pressure due to the gas contained in the partially obstructed intestine, and the mediastinum being greatly displaced to the healthy side, thoracotomy will have slight influence upon the respiration and circulation, especially if intratracheal and insufflation is used. On the other hand, newborn babies resist anoxemia much better than adults; they tolerate anesthetics well, and, above all, the heart at that age presents a marvelous strength. With these facts in mind we did not hesitate to operate upon the baby.

During the operation, twice the respiration stopped and the heart seemed to go into fibrillation. We had a syringe ready with a sixth of 1 c.c. of adrenalin for intracardiac injection, but we were not obliged to use it. Administration of a few whiffs of 10 per cent carbon dioxide and temporary closure of the wound with hot pads each time regulated the heart beatings and the respiration. No temporary block of the phrenic nerve was necessary for a good suture of the diaphragmatic opening.

During the first three days following the operation oxygen and carbon dioxide, 10 per cent mixture, were administered in order to keep a good pulmonary ventilation and to avoid the development of massive or patchy atelectasis, which are the forerunners of postoperative pneumonia and bronchopneumonias. The accumulation of fluid in the chest, which became slightly purulent on the tenth day, was successfully handled by two thoracic taps. A slight infection of both thoracic and abdominal wounds subsided very rapidly.

## SUMMARY

This case is reported because it shows that congenital diaphragmatic hernia can easily be diagnosed if thought of, furthermore that operative treatment is possible, even at a very early age, and that it constitutes the only form of treatment when the intestine is present in the chest cavity and especially when signs of partial intestinal obstruction are present. A careful survey of the literature showed that this is the earliest case of successful operation for diaphragmatic hernia so far reported.

# THE EFFECT OF PULMONARY ARTERY LIGATION ON THE HEALING TIME OF EXPERIMENTAL PYOGENIC LUNG ABSCESSES IN DOGS*

WILLIAM M. TUTTLE, M.D., AND GEORGE L. NICOLL, M.D.
CHICAGO

THE cellular changes which occur following ligation of the pulmonary artery in the normal lung have been rather extensively studied, with widely varying results, since the original experiments of Küttner,[1] Lichtheim[2] and Litten,[3] over a half century ago.

Sauerbruch[4] in 1911 and 1920 reported the ligation of a pulmonary artery in 12 cases of bronchiectasis, with partial success. Two years later Meyer[5] published results in three cases, but the therapeutic value of the procedure was doubtful.

v. Voornveld[6] reported a case each of pulmonary abscess and of bronchiectasis cured by ligation of the pulmonary artery to the involved lobe.

Sauerbruch[7] has tied the pulmonary artery in cases of bronchiectasis and pulmonary lung abscess preliminary to lobectomy, believing that fibrosis and shrinkage of the lung which he noted in animals would also occur in man and act beneficially.

More recently, 1929, Beye[8] ligated the pulmonary artery in two cases of lung abscess to stop hemorrhage, evidently coming from the abscess cavity. One patient died three days postoperatively, the other, while free from hemorrhage, showed no improvement with reference to her pulmonary involvement. There are no published reports on the effects of ligation of the pulmonary artery on the diseased lungs of animals.

## PROCEDURE

Healthy dogs were used in the experiments. All animals were isolated in clean kennels for from one week to ten days before being used. The animals were divided into two groups.

*Group I A.*—Under local anesthesia an embolus infected with staphylococcus aureus was prepared after the method of Cutler, Holman,[9, 10] and their associates, and passed into the circulation by way of the external jugular vein. The embolus which contained a small bird shot, was located immediately by fluoroscopic examination. With aseptic technic and under positive pressure ether anesthesia (Teigel, Meltzer, and Auer[11, 12]) the chest cavity was then opened at the point desired and the artery to the lobe containing the embolus securely tied with black silk, care being taken to avoid injury to the surrounding tissues.

*From the Department of Surgery of the University of Chicago.
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There were five experiments in which the animals were electrocuted on the tenth day.

*Group I B.*—An embolus was prepared and introduced as in Group I A. On the tenth day following embolization, the chest was opened under a procedure similar to that described in Group I A, and the artery to the involved lobe securely tied.

1. In five experiments the animals were electrocuted on the thirty-fourth to thirty-fifth day.

2. In three experiments the animals were sacrificed on the sixtieth to sixty-first day.

3. There were three experiments in which the animals were sacrificed on the ninetieth day.

*Group II A.*—An embolus was prepared and introduced as in Group I. The chest was not opened.

1. Five animals were treated in the same manner described and were killed by electrocution on the tenth day.

2. In six experiments animals were killed on the thirty-fourth to thirty-fifth day.

B. An embolus was prepared and introduced as in the previous groups.

1. In five experiments the chest was opened immediately after embolization and 1 cm. of the pulmonary artery dissected free of adventitia. The artery was not tied. The animals were electrocuted on the tenth day.

2. In seven experiments the pulmonary artery was similarly dissected on the tenth day. Five of these animals were electrocuted on the thirty-fourth and thirty-fifth day and two on the sixty-second day.

*Results.*—Fifty dogs were used in all, thirty-eight of which were actually used to complete these experiments. Causes of death in the twelve dogs that did not survive the experiment were as follows: Distemper, 6; pleuritis following rupture of the abscess, 3; at operation, 1; hemorrhage from abscess, 1; cause unknown, 1.

Wounds as a rule healed per primam; unusual postoperative complications are treated in the protocols. The protocols of one dog from each group appear in this paper. They are typical of the groups.

*Group I A.*—Embolized with a Staphylococcus aureus infected embolus via the external jugular vein and the pulmonary artery to the involved lobe tied on the same day. Sacrificed in ten days.

*Dog 879 B.*—The pleura appeared normal except for a few easily torn adhesions from the left lower lobe to the line of incision. All lobes appeared normal except the left lower, which was increased in size, weight and resistance to pressure. It was dark red to blue black in color. Near the center was an area of fluctuation. The lobe was incised, the cut surface was moist, dark red to blue black in color and at its center was an abscess 2 by 1 by 1.5 cm. filled with purulent and necrotic tissue. The cavity was in communication with a bronchus. Microscopic examination showed an abscess with necrotic walls. There were necrosis and gangrene of

the surrounding lung tissue; no fibroblasts appeared in the abscess wall. Few leucocytes were seen.

*Summary Group I A.*—The abscesses were in all cases large and unhealed; little fibrous tissue appeared in the wall. In some cases no fibroblasts were seen. There was gangrene of the entire lobe in three cases and gangrene and infarction in two.

*Group I B 1.*—Embolized with Staphylococcus aureus infected embolus via the external jugular vein and ligation of the pulmonary artery to the lobe involved on the tenth day. Sacrificed on the thirty-fourth and thirty-sixth days.

*Dog 611 D.*—The pleura appeared normal except along the line of incision where the parietal pleura was fused by fibrous adhesions to the left lower lobe. There was a hard round nodule at the center of the left lower lobe, section through which showed a healed abscess containing the shot. The wall of the small cavity about the shot was white fibrous tissue and the lung tissue distal to this appeared to be compressed. Microscopic examination showed a small cavity with a fibrous tissue wall. There was polymorphonuclear and lymphocytic infiltration into the wall. A wall of leucocytes surrounded the inner wall of the cavity. The bronchi were compressed by the cellular and fibrous tissue infiltration. There was fibrosis of the alveolar walls, the alveoli contained heart failure cells, lymphocytes and a few leucocytes.

*Summary of Group I B 1 (Embolus, Ligation on the Tenth Day).*—The abscesses were healed in all five dogs. A fibrous tissue wall firmly encapsulated the shot. The fibrous tissue in the walls was dense and appeared more marked at the periphery. Polymorphonuclear leucocytes, lymphocytes and heart failure cells were present at the margins of the small cavities left by the bird shot, and in the collapsed lung tissue comprising the inner portion of the wall. Fibrosis appeared in the alveolar walls and in some sections red blood cells appeared in the alveoli.

*Group I B 2.*—Embolized with a Staphylococcus aureus infected embolus. Artery to the involved lobe tied on the tenth day. The animals were sacrificed on the sixtieth and the sixty-first days.

*Dog 667.*—The pleura appeared normal except where it was adherent from the left lower lobe to the line of incision by dense fibrous adhesions. There was a small fistulous tract extending from the left lower lobe. On cut section this abscess measured 2 by 1 by 1 cm. and was situated approximately 2 cm. from the hilus of the lobe. The wall of the cavity was lined with heavy granulation tissue, from which protruded prominent blood vessels. Microscopic examination showed a dense fibrous tissue wall, in which the capillaries were engorged. There were no cells of reaction within the cavity or in the wall.

*Summary of Group I B 2 (Embolized and Artery Tied on Tenth Day).*—Three dogs were used in all and in two of these the abscesses were healed. In the unhealed abscess there was no evidence of an active pyogenic process. Due to the fistula leading away from the cavity and to the nature, size and location of the abscess, one is lead to wonder whether this cavity did not constitute a vestibule for bronchial openings,

thus setting up a chronic bronchial fistula, similar to those resulting from external drainage of lung abscesses in human beings. The healed abscesses showed dense fibrous tissue walls, the collapsed lung tissue being completely replaced by fibroblasts.

*Group I B 3.*—Embolized with a Staphylococcus aureus infected embolus and the artery to the involved lobe tied on the tenth day. Dogs were sacrificed after ninety days.

*Dog 851 B.*—The pleura appeared normal and the left lower lobe was slightly larger than normal, firmer, and deeper red than the remaining lobes. Near the hilus was an area 1.5 by 3 cm. over which the pleura was scarred and below which could be palpated a hard nodular mass. The lung cut with increased resistance and a red frothy fluid appeared on the cut surface. Cut section of the nodule showed it to be a fibrous mass containing at its center the shot. Microscopic examination showed dense fibrosis about the shot, no cells of reaction remained in the wall, there was collapse of the lung tissue surrounding the shot containing cavity.

*Summary Group I B 3.*—In all animals used in this series the abscesses were healed, the wall of the cavity about the shot was dense fibrous tissue, and there was collapse of the surrounding lung tissue with ingrowth of fibroblasts.

*Group II A 1.*—Embolized with Staphylococcus aureus infected embolus via the external jugular vein. Sacrificed in ten days.

*Dog 603 D.*—Dog sacrificed by current. The pleura was smooth and glistening, a few cubic centimeters of clear straw-colored fluid were present in the pleural cavity. All lobes of the lungs appeared normal, with the exception of the left lower lobe, which presented on its posterior aspect near the center, an area 2.5 cm. in diameter, over which the pleura was thickened and the parenchyma a deep gray. The lung tissue through this area cut with some increased resistance, cut section revealing an abscess 9 mm. in diameter, about which the lung tissue was deep red. The abscess cavity was filled with purulent material and contained the bird shot. Microscopic examination showed an acute pyogenic lung abscess; a number of young fibroblasts were present in the collapsed lung tissue composing the abscess walls. Leucocytes and lymphocytes and heart failure cells were seen in the abscess cavity. Bronchitis and infiltration of the surrounding parenchyma with polymorphonuclear leucocytes were noted. A section from below the abscess showed red blood cells within the alveoli.

*Summary of Group II A 1.*—Acute pyogenic lung abscesses were present in six dogs used. One dog died seven days following embolization of hemorrhage from the abscess wall. The abscess wall was composed of collapsed lung tissue; fibroblasts were growing in the outer border of the wall; capillary engorgement, edema, and fibrin formation were present in the collapsed lung tissue forming the wall. Purulent and necrotic tissue filled the cavity. Bronchial communication was present in two dogs and these sections showed bronchitis.

*Group II A 2.*—Embolized with Staphylococcus aureus infected embolus via the external jugular vein. Sacrificed at thirty-four and thirty-five days.

*Dog 856 C.*—The animal was electrocuted on the thirty-fifth day. The pleura was smooth and glistening. A few cubic centimeters of clear straw-colored fluid were present in the pleural cavity. Grossly all lobes appeared normal with the exception of the left lower lobe, which presented a white pleural scar, stellate in configuration, radiating in all directions for a distance of approximately 1.5 cm. Beneath the scar a hard nodule could be palpated.

Section through the area of scarring revealed 0.5 cm. below the surface, a healed lung abscess containing within its fibrous tissue wall the bird shot.

The *microscopic examination* showed a fibrous tissue scar extending downward from the pleura to the abscess wall, fibrous tissue walling off of the shot, anthracosis, atelectasis of the parenchyma about the fibrous tissue wall, blood pigment within the shot-containing cavity, and in the surrounding lung tissue.

*Summary of Group II A 2.*—The abscesses in all six dogs were healed. In two dogs the collapsed lung tissue about the abscess scar contained polymorphonuclear leucocytes. All sections showed fibrous tissue proliferation more marked at the periphery of the wall, the inner wall of the small cavity being composed of collapsed lung tissue, which showed capillary enlargement and in two instances edema.

Hemorrhagic infarction appeared distal to the abscess in three dogs—bronchial communication occurred three times.

*Group II B 1.*—Embolized with infected Staphylococcus aureus embolus, thorocotomy and arterial dissection on same day. Killed in ten days.

*Dog 145 E.*—The pleura was normal, except where the left lower lobe was adherent along the line of thorocotomy incision. In the lower portion of the left lower lobe there was an area which was dark red in color and firmer in consistency than the surrounding lung. Cut section of the lobe revealed a small lung abscess at the upper border of the indurated area. The abscess was filled with purulent material but demonstrated a well-defined wall. Microscopic examination showed acute pyogenic lung abscess.

*Summary of Group II B 1.*—Microscopic examination showed acute pyogenic lung abscesses in all five experiments. Polymorphonuclear leucocytes surrounded the cavities. Distal to the cellular infiltrate the lung tissue was collapsed. Young fibroblasts were growing in toward the center from the periphery.

*Group II B 2.*—Embolized with an infected embolus via the external jugular vein, the chest was opened on the tenth day and the adventitia of the artery to the involved lobe was dissected, but the artery was not tied. Sacrificed on the thirty-fourth, thirty-sixth, and thirty-eighth days.

*Dog 938 D.*—The experiment was terminated on the thirty-fourth day, the animal being electrocuted. The pleura appeared normal except for a few fibrous adhesions extending from the line of incision to the right middle lobe. All lobes appeared normal. On palpation a nodule about the size of a marble was felt in the right lower lobe. The lungs cut with normal resistance and were normal in appearance, with the exception of the nodule, which proved to be a healed lung abscess containing the shot, which was enveloped by a thick wall of fibrous tissue. Microscopic examination showed a small circumscribed cavity which was in communication with a

bronchus, the epithelium of which did not grow down into the cavity. Fibroblasts were present in the wall, being in greater abundance at the periphery, where they encapsulated small intramural abscesses. Edema and fibrosis were present in the alveolar walls. The pleura over the healed abscess was thickened.

*Summary of Group II B 2.*—The abscesses in all five dogs were healed. Fibrosis was present in the walls of the small cavity which contained the shot, being more marked at the periphery. The walls appeared to be composed of collapsed lung tissue into which had grown fibroblasts. Bronchial communication developed in one animal. Pleural thickening occurred over the abscess site. The alveolar walls were slightly thickened by the ingrowth of fibrous tissue and small amounts of old blood appeared in the alveoli.

TABLE I

EMBOLIC INOCULATION WITH STAPHYLOCOCCUS AUREUS

| DAYS | 7 | 10 | 34 and 35 |
|---|---|---|---|
| RESULTS | 1 experiment. Large abscess. Hemorrhage from cavity. | 5 experiments. All abscesses unhealed. Hemorrhagic infaret distal to abscess in 3 cases. | 5 experiments. All abscesses healed. Fibrous tissue encapsulating shot. |

TABLE II

EMBOLIC INOCULATION WITH STAPHYLOCOCCUS AUREUS, THOROCOTOMY, ARTERY TO INVOLVED LOBE TIED ON TENTH DAY FOLLOWING EMBOLIZATION

| DAYS | 34 and 36 | 60 and 61 | 90 |
|---|---|---|---|
| RESULTS | 5 experiments. All abscesses healed. | 3 experiments. Abscesses healed. 1 large abscess unhealed. No cells of reaction present. Fibrous tissue more marked than 34 to 35 days. | 3 experiments. All healed abscesses. Alveolar thickening. |

*Group II B 3.*—Embolized with a Staphylococcus aureus infected embolus. The adventitia of the artery to the involved lobe dissected free on the tenth day, but the artery was not tied. Animals sacrificed on the sixty-first day.

*Dog 997 B.*—The upper left lobe was slightly adherent along the line of incision, the pleura appeared normal throughout the remainder of the chest. At the lower border of the left upper lobe was a hard fibrotic area measuring 1 by 1 cm. On cut section this nodule revealed a healed lung abscess with the shot at the center. Microscopic examination showed a dense wall of fibroblasts surrounding the shot cavity, which contained a few heart failure cells and lymphocytes.

*Summary of Group II B 3 (Embolized, Adventitia Freed, Artery Not Tied).*—The abscesses were healed in all cases, the wall of fibroblasts was dense and there was slight thickening of the alveolar septi.

TABLE III

EMBOLIC INOCULATION WITH STAPHYLOCOCCUS AUREUS. ARTERY TO INVOLVED LOBE LIGATED ON THE SAME DAY

| DAYS | 1 | 7 | 10 |
|---|---|---|---|
| RESULTS | 1 experiment. No abscess. | 1 experiment. Large abscess. Rupture into pleural cavity. Pleuritis. | 4 experiments. All abscesses large. Gangrene of lobe occurred 2 times. Hemorrhagic infarction with necrosis of alveolar walls in two animals. |

TABLE IV

EMBOLIC INOCULATION WITH STAPHYLOCOCCUS AUREUS, THOROCOTOMY, ARTERIAL DISSECTION ON TENTH DAY

| DAYS | 34 and 36 | 60 |
|---|---|---|
| RESULTS | 5 experiments. All animals showed healed abscesses. | 2 experiments. Healed abscesses in both cases. |

TABLE V

EMBOLIC INOCULATION WITH STAPHYLOCOCCUS AUREUS, THOROCOTOMY AND ARTERIAL DISSECTION ON THE SAME DAY

| DAYS | 10 |
|---|---|
| RESULTS | All abscesses unhealed, medium in size (0.5-1 cm.) pyogenic wall well formed. |

## DISCUSSION

It appears from the foregoing experiments that healing of pyogenic lung abscesses in the dog is not markedly altered by ligation of the pulmonary artery when the artery is tied after the abscess is encapsulated.

Ligation of the artery on the same day as embolization results in hemorrhagic infarction of the entire lobe and in some instances in gangrene. There was in every case a large abscess with necrotic walls showing little evidence of a pyogenic membrane in the infarcted or gangrenous lobe.

The average time for spontaneous healing of experimental pyogenic lung abscesses in the dog has been shown by Van Allen, Adams and Hrdina[16] to be in the neighborhood of thirty to thirty-five days. Despite the fact that in all animals used in this work the infected embolus contained a lead shot, thus constituting a foreign body in the tissues, all abscesses in both the dissected and ligated series were healed when the dogs were sacrificed not earlier than thirty-five days, with the exception of Dog 783 B which remained unhealed at the end of two months.

The microscopic picture was almost identical in both the ligated and dissected series and differed from the nonthoracotomized group (Group II A 2) in that the latter showed more cellular response in the abscess wall.

The lungs of the animals which were allowed to run for a longer period (two to three months) showed more fibrosis about the shot and in the adjacent parenchyma and all signs of cellular reaction had vanished.

The work of Reichert, Holman and Mathes[13, 15] has shown that in experimental lung abscess in dogs the branches of the bronchial artery about the abscess undergo marked dilatation, when the pulmonary artery to the entire lobe is not embarrassed. This may be offered as an explanation as to why the abscesses developed so rapidly and extensively and the surrounding lung tissue showed such marked infarctive and gangrenous change in the animals in which the pulmonary artery to the entire lobe was tied on the day of embolization (Group I A) as contrasted to the almost negligible disturbance of the healing time in those ligated on the tenth day. In the former group the bronchial artery had not had sufficient time to dilate. Hence, ligation of the pulmonary artery was more disastrous.

Bronchial artery dilatation about the abscess site may also explain why, in spite of an infected area within the lobe, hemorrhagic infarction and gangrene did not occur about the abscess in that group in which the pulmonary artery was ligated ten days following embolization, although in several cases infarction was seen distal to the abscess. Infarction distal to the abscess undoubtedly took place at the time of embolization, for it was seen in both Group II A 1 and II A 2 in neither of which the chest had been opened. Fibrosis of the alveolar walls and fibrous tissue septi was not as marked in the unaffected portions of the lobe as was the case when the pulmonary artery to a normal lobe was ligated, in work reported previously by Van Allen, Nicoll and Tuttle.[17] The phenomena of bronchial artery dilatation may also account for the absence of gangrene in Group I B and its common occurrence following pulmonary artery ligation in pneumonic lungs, and following ligation of the artery to incised normal lungs as reported by Olch and Ballon.[14] Evidently, the dilated bronchial artery, with its correspondingly increased blood capacity guards the infected lung tissue from gangrene when the pulmonary artery is tied.

These experimental results would seem to militate against the use of pulmonary artery ligation as a pure therapeutic procedure and especially in those cases where unrecognized early abscesses might be present.

## CONCLUSIONS

1. Ligation of the pulmonary artery in the dog has no effect on the healing time of a pyogenic abscess after encapsulation has started.

2. Ligation of the pulmonary artery in the dog has a markedly detrimental effect on a beginning lung abscess increasing the extent of involvement, limiting pyogenic membrane formation and sometimes causing gangrene of the lobe.

REFERENCES

1. Küttner: Virchows Arch. **73**: 476, 1878.
2. Lichtheim: Arch. f. exp. Path. **10**: 54, 1878.
3. Litten, M.: Ztschr. f. klin. Med. **1**: 131, 1880.
4. Sauerbruch, F.: quoted from Schlaepfer, K.: Arch. Surg. **9**: 25, 1924.
5. Meyer, W.: Ann. Surg. **58**: 188, 1913.
6. v. Voornveld: quoted from Schlaepfer, K.: Arch. Surg. **9**: 25, 1924.
7. Sauerbruch, F.: Arch. f. klin. Chir. **148**: 721, 1927.
8. Beye, H.: Arch. Surg. **18**: (pt. 2) 520, 1929.
9. Cutler, E. C., and Schleuter, S. A.: Ann. Surg. **84**: 256, 1926.
10. Holman, E., Weidlein, I. F., Schleuter, S. A.: Proc. Soc. Exp. Biol. and Med. **23**: 266, 1926.
11. Teigel, M.: Mitt. a. d. Grenzgeb. d. Med. u. Chir. **3**: 789, 1907.
12. Meltzer, S., and Auer, J.: J. Exper. Med. **11**: 622, 1909.
13. Reichert, Holman, and Mathes: Proc. Soc. Exp. Biol. and Med. **27**: 278, 1930.
14. Olch and Ballon: Arch. Surg. **19**: 1586-1594, pt. 2, 1929.
15. Holman, E.: Arch. Otolaryng. **11**: 287, 1930.
16. Van Allen, Adams and Hrdina: Arch. Surg. **19**: 1262, pt. 2, 1929
17. Van Allen, Nicoll and Tuttle: Yale J. Biol. and Med., May, 1930.

# THE EFFECTS OF COMPLETE OCCLUSION OF THE THORACIC AORTA. AN EXPERIMENTAL STUDY*

ALFRED BLALOCK, M.D.
NASHVILLE, TENN.

SEVERAL investigators have been interested in the effects of the occlusion of the thoracic aorta. Erlanger and Gasser[1] employed this procedure as a means of producing shock. In experiments on three dogs, the thoracic aorta was partially occluded for two hours. Artificial respiration was used throughout the experiments. Partial occlusion of the aorta was followed by a rise in the carotid blood pressure which returned to normal, or slightly below normal, at the end of the two-hour occlusion period. The blood pressure declined rapidly following the release of the occlusion and death resulted. Burton-Opitz[2] studied the effects of occlusion of the thoracic aorta on the amount of blood that flowed through the superior and inferior venae cavae. Immediately following the occlusion he found an increase in the amount of blood that flowed through the superior vena cava and a decrease in that through the inferior cava. Barcroft[3] measured the systemic output of the heart of dogs with a mechanical stromuhr. Complete occlusion of the thoracic aorta was found to increase the cardiac output if the vagi were sectioned. Conflicting results were obtained when the vagus nerves were not divided. The occlusion was maintained for only a few minutes. The increase in the output of the heart was shown to be due to the transference of blood from the vessels supplied by the thoracic aorta to the remainder of the vascular system.

Reid[4] has recently reported two instances of occlusion of the thoracic aorta in patients for aneurysm of the abdominal aorta. One of these was operated upon by Reid and the other by Andrus. In one instance the thoracic aorta was occluded by a ball of fascia that was held in place by mattress sutures of heavy silk. In the other, the aorta was doubly ligated and divided. During the course of both operations, it was observed that the radial pulse became stronger as soon as the aorta was occluded. Reid's patient died twelve hours following the operation. "At autopsy, it was discovered that death had resulted from hemorrhage that came from the divided intercostal artery at the posterior part of our incision into the chest wall." The other patient died one and one-half hours after the completion of the operation. Although the operation was performed extrapleurally, 400 c.c. of blood were found in the right pleural cavity at autopsy.

*From the Department of Surgery of Vanderbilt University.
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The present experiments were performed primarily in order to determine the effects of complete occlusion of the thoracic aorta on the output of the heart. The only similar studies are those of Barcroft and he determined only the initial alterations. The experiments of Barcroft and of Erlanger and Gasser were performed with the chest open and artificial respiration was employed.

## METHODS

Dogs were used in all experiments. One grain of morphine was given an hour or more before the control determinations were performed. Following the control studies, the operation was carried out under positive pressure intratracheal ether anesthesia. An intercostal incision was made in the eighth interspace on the left side and the pleural cavity was entered. In two experiments, the thoracic aorta was ligated doubly. After inflating the lungs, the incision in the chest wall was closed tightly. Additional determinations were performed after allowing a sufficient time for the animals to recover from the ether anesthesia which in no instance was profound. In two experiments, a heavy piece of cord that was not tied was placed around the aorta and its ends were brought out through the incision. After closing the incision in the chest, studies were performed with the aorta patent. The cord was then twisted until the femoral pulsation was obliterated and additional studies were carried out.

The oxygen consumption was determined by a Benedict spirometer with a graphic recording device. Samples of blood for the gas analyses were withdrawn under oil from the right and left ventricles of the heart. The oxygen and carbon dioxide contents of the arterial and venous blood were determined by the use of the Van Slyke-Neill manometric apparatus. The output of the heart was calculated from the Fick formula.

$$\frac{\text{C.c. } O_2 \text{ consumed per minute}}{\text{Amt. } O_2 \text{ taken up in lungs by 1 c.c. of blood}} = \text{Number of c.c. of blood flowing through the lungs per minute.}$$

In two additional experiments, no studies were performed other than simply determining the survival period of the dogs following ligation of the thoracic aorta.

## RESULTS

The operative procedure of opening and closing the chest without occlusion of the thoracic aorta did not result in a decrease in the output of the heart or in the consumption of oxygen. Studies on the output of the heart were not performed earlier than forty-five minutes following the occlusion of the aorta. The animals appeared to be in very good condition for an hour following the ligation except for the inability to use the posterior extremities. There was an increase in pulse rate and a decline in rectal temperature in all experiments. The oxygen consumption began to decrease immediately following the occlusion. In one instance it declined to 28 per cent of the original figure at the end of two hours, but usually the fall was not so great. A graphic record of the alterations in the consumption of oxygen that are produced by occlusion and de-occlusion of the aorta are given in Figs. 1 and 2.

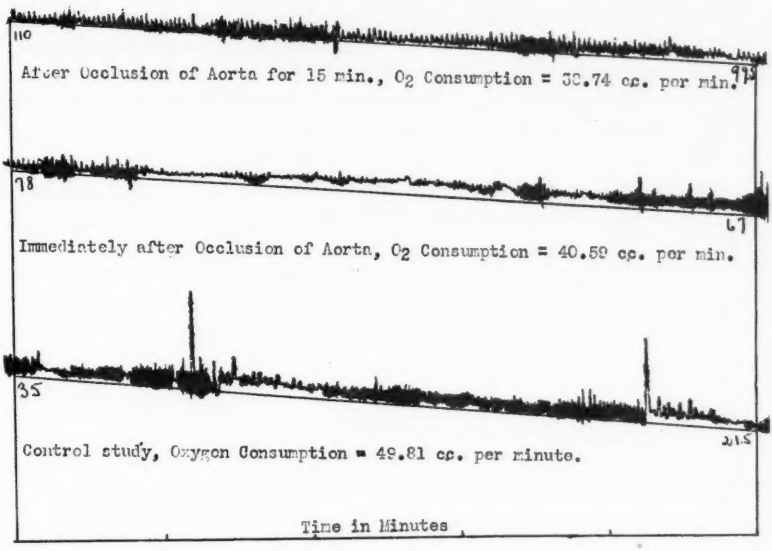
110
After Occlusion of Aorta for 15 min., O2 Consumption = 30.74 cc. per min.
78
Immediately after Occlusion of Aorta, O2 Consumption = 40.59 cc. per min.
35
Control study, Oxygen Consumption = 49.81 cc. per minute.
Time in Minutes

Fig. 1.—The effect of complete occlusion of thoracic aorta on $O_2$ consumption.

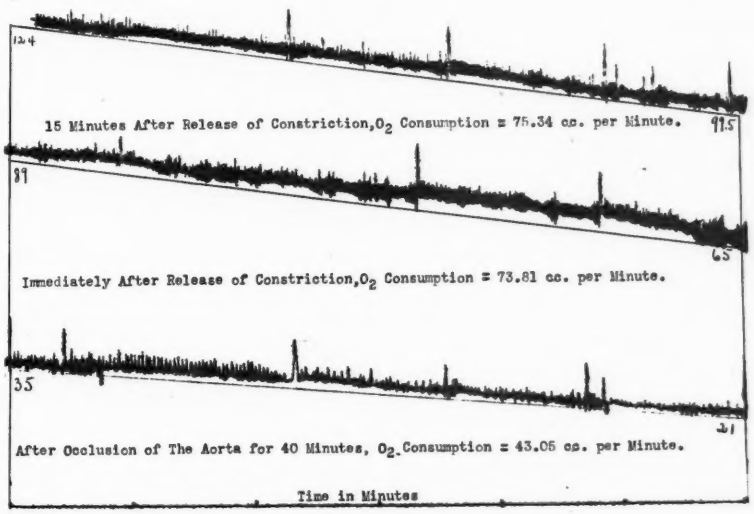
124
15 Minutes After Release of Constriction, O2 Consumption = 75.34 cc. per Minute.
89
Immediately After Release of Constriction, O2 Consumption = 73.81 cc. per Minute.
35
After Occlusion of The Aorta for 40 Minutes, O2 Consumption = 43.05 cc. per Minute.
Time in Minutes

Fig. 2.—The effect of releasing occlusion of the thoracic aorta on the oxygen consumption.

Very little change in the oxygen content of the arterial blood was found following the occlusion. The oxygen content of the mixed venous blood diminished markedly. There was a great decrease in the carbon dioxide content of both the arterial and mixed venous blood in the one experiment in which it was determined. The output of the heart per minute and per beat decreased markedly in all experiments. The results of these experiments are given in Tables I, II, III, and IV.

No free fluid was found in the pleural or peritoneal cavities at autopsy. The liver was very hard. The small vessels of the intestines appeared very prominent.

As has been stated, two experiments were performed in which the output of the heart was not determined and simply the survival period was noted. One of these animals lived three hours following the occlusion

TABLE I

THE EFFECTS OF THE LIGATION OF THE THORACIC AORTA

| TIME | WEIGHT KG. | PULSE RATE PER MIN. | ARTERIAL OXYGEN VOLS. % | VENOUS OXYGEN VOLS. % | A. V. DIF. VOLS. % | OXYGEN CONSUMPTION, C.C. PER MIN. | CARDIAC OUTPUT C.C. PER MIN. | OUTPUT PER BEAT C.C. |
|---|---|---|---|---|---|---|---|---|
| 7:00 P.M. Control study | 5.9 | 71 | 17.10 | 9.44 | 7.66 | 58.1 | 759 | 10.7 |
| 11:00 P.M. 45 min. after ligation | 5.9 | 180 | 15.14 | 5.56 | 9.58 | 23.06 | 241 | 1.3 |
| 12:00 midnight 2 hours after ligation | 5.9 | 185 | 13.61 | | | 16.6 | | |

*Protocol:* 6:00 P.M., one grain of morphine. 7:00 P.M., tracheotomy tube placed in, control studies performed. 10.00 P.M., ether anesthesia, chest opened, thoracic aorta ligated twice, time consumed 20 minutes. 11:00 P.M., animal conscious, reflexes present, no râles heard in chest, heart beat forceful, slight engorgement of veins of abdomen. 12:00 midnight, animal appeared much sicker. 12:20 A.M., animal died. Autopsy revealed much venous engorgement of the abdominal viscera. Lungs appeared normal. Right heart seemed to be dilated. Much clear fluid poured from the nose and mouth.

and the other three hours and fifteen minutes. In another experiment the abdominal aorta was ligated just below the diaphragm and above the coeliac axis. Death resulted three and one half hours following the operation.

## DISCUSSION

Erlanger and Gasser found that partial occlusion of the aorta and partial occlusion of the vena cava resulted in similar changes. Concerning the results of the experiments in which the aorta was occluded, they state, "The fall of the arterial pressure to the level of 50 mm. Hg is not due to inefficiency of the vasomotor mechanism, either central or peripheral, nor to inefficiency of the heart; it must, therefore, find its

TABLE II

THE EFFECTS OF LIGATION OF THE THORACIC AORTA

| TIME | WEIGHT KG. | RECTAL TEMPERATURE FAHRENHEIT | PULSE RATE PER MIN. | ARTERIAL OXYGEN VOLS. % | VENOUS OXYGEN VOLS. % | A. V. DIF. VOLS. % | OXYGEN CAPACITY VOLS. % | % SATURATION | OXYGEN CONSUMPTION C.C. PER MIN. | CARDIAC OUTPUT C.C. PER MIN. | OUTPUT PER BEAT C.C. |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 5:30 P.M. Control Study | 9 | 97 | 85 | 16.68 | 12.32 | 4.36 | 17.43 | 96 | 59.04 | 1354 | 16 |
| 7:45 P.M. 1 hour after ligation | 9 | 96 | 92 | 17.60 | | | 17.99 | 98 | | | |
| 8:15 P.M. 1½ hours after ligation | 9 | 95½ | 170(?) | 15.95 | 4.68 | 11.27 | | | 44.3 | 392 | 2.3 |

*Protocol:* 4:00 P.M., one grain of morphine. 5:00 P.M., one-half grain of morphine. 5:10, tracheotomy. 5:25 P.M., control study. 6:45 P.M., aorta ligated, tracheal insufflation, very small amount of ether being used. 7:45 P.M., animal appeared to be in good condition. Venous sample drawn at this time clotted. 8:15 P.M., studies performed. 9:00 P.M., animal died. Much venous congestion of all of abdominal viscera. Lungs appeared normal.

TABLE III

THE EFFECTS OF OCCLUSION OF THE THORACIC AORTA

| TIME | WEIGHT KG. | PULSE RATE PER MIN. | ARTERIAL OXYGEN VOLS. % | VENOUS OXYGEN VOLS. % | A. V. DIF. VOLS. % | OXYGEN CAPACITY VOLS. % | % SATURATION | OXYGEN CONSUMPTION C.C. PER MIN. | CARDIAC OUTPUT C.C. PER MIN. | OUTPUT PER BEAT C.C. |
|---|---|---|---|---|---|---|---|---|---|---|
| 5:20 P.M. 25 min. after closing chest | 7.3 | 48 | 20.27 | 15.38 | 4.89 | 20.87 | 97.1 | 59.04 | 1207 | 25.2 |
| 6:30 P.M. 95 min. after closing chest | 7.3 | 50 | 20.30 | 14.9 | 5.4 | | | 55.35 | 1025 | 20.5 |
| 8:00 P.M. 50 min. after occluding aorta | 7.3 | 48 | 20.87 | 10.25 | 11.62 | 21.47 | 97.2 | 38.74 | 334 | 7.0 |
| 10:00 P.M. 2 hr. after constriction released | 7.3 | 165 | 21.47 | 7.51 | 13.96 | | | 47.97 | 344 | 2.1 |
| 1:00 A.M. 5 hr. after constriction released | 7.3 | 150 | 21.60 | 9.80 | 11.80 | 22.54 | 96 | 46.12 | 391 | 2.6 |

*Protocol:* 3:30 P.M., one gr. morphine. 4:40 P.M., chest opened, ligature placed around thoracic aorta but not tied. Chest closed tightly. Operation completed at 4:55 P.M. 5:10 P.M., studies made with ligature untied. 6:30 P.M., another study made with ligature untied. 7:10 P.M., ligature around the aorta twisted, obliterating the femoral pulsation. 8:00 P.M., first studies made with thoracic aorta occluded. 8:10 P.M., occlusion of aorta released by untwisting the heavy ligature around it. Immediate increase in pulse rate. 10:00 P.M., first studies made with constriction released. Dog seemed to be in fair condition. 1:00 A.M., second studies made with constriction released. Study was abandoned at this time and dog found dead 6 hours later. Reddening of the abdominal viscera was found at autopsy. There was no free fluid in pericardial or pleural cavities. It was seen at this time that the lumen of the aorta could be easily obliterated by twisting the cord which had been placed around it.

TABLE IV

THE EFFECTS OF OCCLUSION OF THE THORACIC AORTA

| TIME | WEIGHT KG. | PULSE RATE PER MIN. | TEMPERATURE F. RECTAL | ARTERIAL $O_2$ VOLS. % | VENOUS $O_2$ VOLS. % | A. V. DIF. VOLS. % | ARTERIAL $CO_2$ VOLS. % | VENOUS $CO_2$ VOLS. % | $O_2$ CONSUMPTION C.C. PER MIN. | CARDIAC OUTPUT PER MIN. C.C. | OUTPUT PER BEAT C.C. |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 6:30 P.M. Control Study | 8.7 | 40 | 98 | 17.31 | 11.38 | 5.93 | 46.43 | 51.29 | 57.6 | 971 | 24 |
| 9:10 P.M., one hr. after opening and closing chest. | 8.7 | 80 | 98 | 16.58 | 10.30 | 6.28 | 47.80 | 49.21 | 62.5 | 995 | 12 |
| 11:10 P.M., one hr. after completely occluding the aorta. | 8.7 | 180 | 95 | 16.35 | 5.21 | 11.14 | 24.19 | 30.72 | 23.4 | 205 | 1.1 |

*Protocol:* 5:30 P.M., one grain of morphine. 6:30 P.M., control studies. 8:00 P.M., under light ether anesthetic and intratracheal insufflation, chest opened, ligature placed around thoracic aorta (not tied) and chest closed. 9:10 P.M., studies with ligature in place but not tied. 10:10 P.M., cord around aorta twisted until peripheral pulsation was obliterated. 11:10 P.M., studies with aorta occluded. The femoral artery was then cut across and there was only a very slow flow of blood from its central cut end.

explanation in a reduction in the effective volume of blood." The fall in arterial pressure recorded by Erlanger and Gasser and the marked reduction in the cardiac output reported in the present paper would seem to indicate that the heart is not being overtaxed. Erlanger and Gasser found that the peripheral resistance was practically normal at the end of the occlusion period and hence concluded that inefficiency of the vasomotor system was not the cause for the decline in arterial pressure. That occlusion of the aorta works injuriously to the circulation by damaging the parts in which the flow of blood is diminished is suggested by the further drop in arterial blood pressure after de-occlusion in the work of Erlanger and Gasser and the failure of the cardiac output to increase in our experiments.

The findings of Barcroft are not comparable to those reported here because his observations were made immediately following the occlusion of the aorta and an interval of at least forty-five minutes separated the occlusion and the first studies in our experiments. The early increase in the carotid arterial pressure following aortic occlusion is followed in a short time by a decline. It is likely that the initial increase in the output of the heart is sustained for only a few minutes.

The present experiments were performed on normal dogs. It has been proved[2] that anastomotic channels carry some blood to the posterior part of the body of experimental animals after occlusion of the thoracic aorta. The lower the ligation is performed, the greater are the number of such channels. Halsted[5] produced partial occlusion of the thoracic aorta of dogs by the application of metal bands. Some animals remained alive in which the lumen of the artery was greatly reduced but in no instance was the occlusion complete. We cannot state what the results would have been in the present experiments had dogs been used which had previous abnormalities of the circulatory system, such as aneurysm of the abdominal aorta. It is possible but extremely unlikely that an aneurysm of the abdominal aorta would result in the formation of a collateral circulation that was sufficient to allow the occlusion of the thoracic aorta without resulting in death. It is especially difficult to believe that organs such as the kidneys and adrenals could receive enough blood supply by collateral branches.

REFERENCES

1. Erlanger, J., and Gasser, H. S.: Studies in Secondary Traumatic Shock. II. Shock Due to Mechanical Limitation of Blood Flow, Am. J. Physiol. **49**: 151, 1919.
2. Burton-Opitz, R.: The Venous Supply of the Heart, J. Physiol. **58**: 253, 1921.
3. Barcroft, Henry: Cardiac Output and Blood Distribution, J. Physiol. **71**: 280, 1931.
4. Reid, Mont R.: A Report of Vascular Lesions Which Illustrate Some of the Fundamental Principles that Have Been Taught and Emphasized by Professor Rudolph Matas, Am. J. Surg. **14**: 17, 1931.
5. Halsted, W. S.: Partial, Progressive and Complete Occlusion of the Aorta and Other Large Arteries in the Dog by Means of the Metal Band, J. Exper. Med. **11**: 373, 1909.

# MEDIASTINOTOMY FOR EXPERIMENTS ON THE HEART AND LUNGS IN THE DOG*

CYRUS F. HORINE, M.D.
BALTIMORE, MD.

IT IS difficult to expose the heart and the great vessels of the lungs in the dog without producing a pneumothorax, and therefore, positive pressure anesthesia is required in doing many experiments on the heart and lungs. We wish to present a method for doing certain problems in which the question of pneumothorax has been eliminated. The results

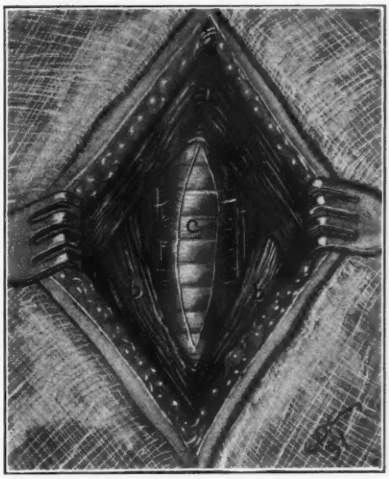

Fig. 1.—Incision through the skin and subcutaneous tissues. *A*, the sternomastoid group of muscles; *B*, the sternothyroid group of muscles; *C*, the exposed trachea, after an incision has been made through the fascia.

obtained in other problems, by the use of this method, will be published elsewhere.

The best type of dog to be used in this work is a beagle, or any dog having a short thick chest. The choice of anesthetic depends upon the problem to be proved.

An incision is made in the midline of the neck extending from the sternum to the larynx. The subcutaneous tissues are exposed and incised. The sternothyroid group of muscles are separated and the trachea comes into view. A broad retractor with a blade about one and one-half

*From the Department of Surgery, School of Medicine, University of Maryland.
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inches in length, is then placed between the separated muscles and applied with pressure under the upper end of the sternum. This procedure produces tension upon the areolar tissue covering the trachea and innominate vessels. This connective tissue is then punctured exactly in the midline over the trachea with a plunt pointed clamp. The clamp passes under the innominate vessels. The index finger can then be used for blunt dissection to enlarge the opening. A longer retractor containing a light replaces the first retractor. This retractor elevates the innominate and subclavian vessels and gives a deeper exposure between the two pleural cavities immediately over the trachea. Blunt dissection is then carried on with a long clamp containing a small gauze sponge.

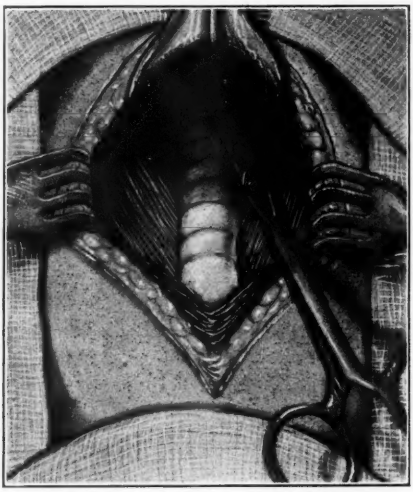

Fig. 2.—After the trachea has been exposed a retractor has been introduced beneath the sternomastoid muscles and the fascia which covers the trachea. The clamp is used to start the dissection beneath the innominate vessels.

Gentle forward and lateral manipulation with a clamp containing a sponge will dissect the areolar tissue over the trachea and bring into view the bifurcation of the trachea, the base of the heart, the ascending and descending aorta, the vena azygos and the superior vena cava. If the vena azygos is retracted to the right, the right branch of the pulmonary artery can be seen entering the lung. If the aorta is retracted to the left, the left branch of the pulmonary artery is shown entering the left lung. A group of mediastinal glands is found to the right of the aorta in front of the left pulmonary artery.

The pulmonary veins are difficult to expose to any great degree because of the possibility of injury to the pleura. However, they are found to be posterior to the pulmonary arteries.

The greater portion of the esophagus is exposed in this operation. If it becomes distended with air, the peristalsis of the esophagus, with each respiratory excursion may obstruct the field of vision. A small rubber tube inserted into the esophagus will overcome this difficulty.

The above-mentioned exposures may be made with the pressures within the pleural cavities remaining the same as before operation. The development of a pneumothorax will cause an immediate bulging of the pleura toward the midline.

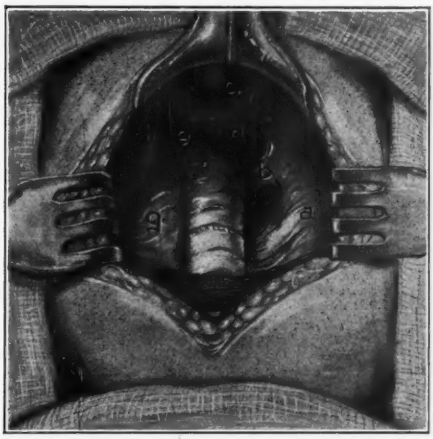

Fig. 3.—In this illustration the dissection has been carried to the base of the heart after a longer retractor has been introduced into the wound. This retractor contains a small light not shown in the diagram. The dissection has been carried to the depth of the wound by using a sponge on a long forceps.

*A*, the superior vena cava; *B*, the vena azygos; *C*, the right auricle; *D*, the right pulmonary artery; *E*, the left pulmonary artery, and *F*, the ascending and descending aorta; *G*, the esophagus.

The pulmonary arteries are covered with a thick fibrous fascia. They may be completely exposed if this fascia is carefully incised. This may be done without producing a pneumopericardium. A ligature may be passed around the vessels by using an aneurysm needle.

If the operation is done under aseptic technic, healing will occur in the majority of cases without infection. The wounds heal with very little scar formation.

# DISTRIBUTION OF THE PULMONARY AND BRONCHIAL CIRCULATION*

## (An Experimental Study)

Cyrus F. Horine, M.D., and C. Gardner Warner, M.D.
Baltimore, Maryland

Virchow, R.,[1] believed in the exchange of bloods in the pulmonary and bronchial systems. Küttner,[2] in 1874, suggested that there is a free interchange of blood with rich anastomosis between the bronchial and pulmonary vessels in the frog's lung. Cohnheim[3] and Litten[4] in their earlier writings denied the existence of anastomosis of these vessels but later admitted that such anastomosis existed. Haller,[17] Hyrtl,[18] Henle[19] and Luschka[20] maintained that free anastomosis exists. Konigstein[5] in a study of comparative anatomy and physiology in lungs of reptiles, birds, and amphibians reached the same conclusion. Further work upon this subject has been carried out by Tigerstedt,[6] Zuckerkandl,[7] Miller,[11, 12, 13, 14, 15, 16] Ghoreyeb, A. A., and Karsner, H. T.,[8] and Karsner and Ash.[9]

Coryllos, Pol N., and Birnbaum, G. L.,[10] quote the work of William Snow Miller[14, 16] as follows: "Miller in a series of papers showed that the bronchial artery distributed itself along the entire length of the bronchi giving small branches which form capillary plexi in the submucosa of the bronchi and bronchioli (as far as the respiratory bronchioli and ducti alveolaris) around the bronchial glands and even in the septums between the alveoli. At almost regular intervals these capillaries join venous radicals which are situated in the mucosa. The venous radicals form a rectangular plexus corresponding to the long axis of the bronchi. From this venous plexus, branches pass through the muscular layer and form a second plexus which is situated along the boundary line between fibrosa and muscularis. The second plexus is the source of venous radicals which in the deeper part of the lung form one of the sources of the pulmonary vein, these correspond to the bronchopulmonary vein of Le Fort. On the other hand, the venous radicals which arise from the first two or three divisions of the bronchi do not join the pulmonary vein but form the true bronchial veins which empty into the azygi, the intercostal veins, or the superior vena cava. The important point is that although there are no anastomoses between bronchial arteries and pulmonary arteries, the bronchial capillaries, partly at least, communicate with the pulmonary vein system through the bronchopulmonary veins of Le Fort."

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*From the Departments of Surgery and Pathology, University of Maryland, School of Medicine.
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Miller, working with dead animals, injected Berlin blue gelatin into the pulmonary artery at a pressure of 112 mm. of mercury with the pulmonary vein open and found that the bronchial vessels were not injected. The bronchial vessels were partially injected when the gelatin was introduced into the pulmonary vein with the pulmonary artery open and the bronchial vessels also became partially injected if the gelatin was introduced into the pulmonary artery with the pulmonary vein clamped. The bronchial vessels became completely injected, if the gelatin was injected into the pulmonary vein with the pulmonary artery being clamped. He therefore concluded that there is no anastomosis between bronchial arteries and pulmonary arteries. Miller then injected Berlin blue in 8 per cent gelatin into the pulmonary vein until it flowed freely from the pulmonary artery. The pulmonary vein was clamped and 8 per cent vermilion granules in gelatin was forced into the pulmonary artery. The pulmonary artery showed red, while the bronchial vessels and the pulmonary vein showed blue. He concluded that an anastomosis exists between the capillaries of the bronchial arteries and the capillaries of the pulmonary veins.

Wishing to study the circulation of the lung under conditions approaching the normal, injections were made into the bronchial and pulmonary systems using different colored materials in the two systems. The work was carried out on living animals without pneumothorax. Thirty-five dogs were used in these experiments.

## METHOD

An opening into the mediastinum of the dog was made through a longitudinal incision over the trachea. The dissection was carried down to the base of the heart between the pleural sacs. The ascending aorta and right pulmonary artery were exposed.* A cannula was placed in the left carotid artery. The dog was bled from this cannula to reduce the volume of blood. Higgins' concentrated stock black India ink was used in bronchial system. A suspension of finely divided particles of carmine red in glycerine and formalin was used in the pulmonary system. In this same suspension coarsely divided particles of viridine green was used. The particles of last named color were of such size that they could not pass through the pulmonary capillaries. These large particles were of value in differentiating between the larger radicals of the pulmonary artery and vein. The black ink was injected through the cannula in the left carotid artery at a pressure of 150 mm. of mercury, while a simultaneous injection of the carmine red and viridine green at a pressure of 30 mm. of mercury was injected into the right pulmonary artery. The injection into the right pulmonary artery was made with a syringe equipped with a long needle. Simultaneously with the beginning of the

*This method of approach devised by one of us (C. H.) is published in detail in THE JOURNAL OF THORACIC SURGERY 2: 77, 1932.

injections into the two systems the ascending aorta and the main pulmonary artery were clamped near the base of the heart with the same clamp, a long Rochester pean clamp introduced also through the opening in the mediastinum. Another clamp was applied to the trachea to stop respirations and to prevent collapse of the lungs. Ten per cent formalin solution was injected into the trachea to fix the lungs before removal from the chest cavity.

The black ink, therefore, was injected into the bronchial system and the carmine red and viridine green were introduced directly into the right pulmonary artery. The formalin in the suspension injected into

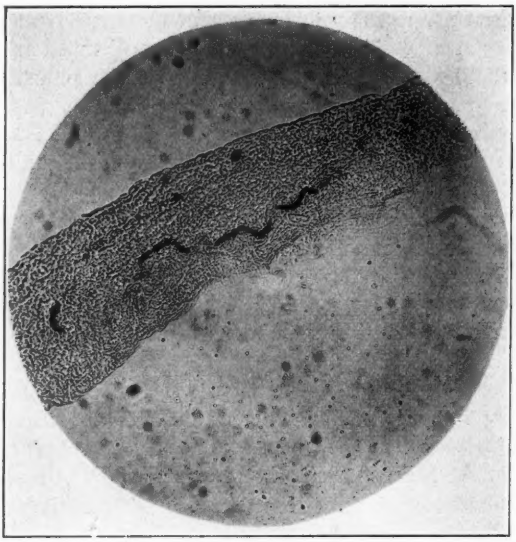

Fig. 1.—The unstained microphotograph of a thick frozen section (20 microns) of the pulmonary ligament shows the injected material in the bronchial arteries, magnification approximately ×200. The specimen was taken from the pleural ligament near its reflection over the lower right lobe.

the pulmonary artery aided in the early fixation of the tissues. The injections into both systems with the clamping of the aorta, pulmonary artery and trachea required from three to four seconds in time.

We have made a study of thirty-five specimens of injected lungs. Frozen and permanent sections of lungs were examined with the binocular microscope using the stereoscopic eyepieces. Gross examination of the right lungs showed a good injection with the black ink in the vessels around the hilum and in the parietal and visceral pleura. Bronchial vessels could be traced to the visceral pleura. This was especially true over the lower lobe where the injected vessels were larger. Dissections of the mediastinal vessels were made in five dogs. An anastomosis was

found to exist between the bronchials, the internal mammaries, the intercostals and the esophageals. Histologic examinations of specimens in all cases showed black particles in the visceral pleura. This, together with the findings in gross dissections, shows without doubt that the visceral pleura of the dog received blood from arterial radicals, in part at least from the bronchials, which is contrary to Miller's findings.

Microscopic examination of the pulmonary vessels showed that none of them contained a mixture of black, red and green particles. The green particles which had been introduced into the right pulmonary artery could not pass through the capillary bed and therefore had been

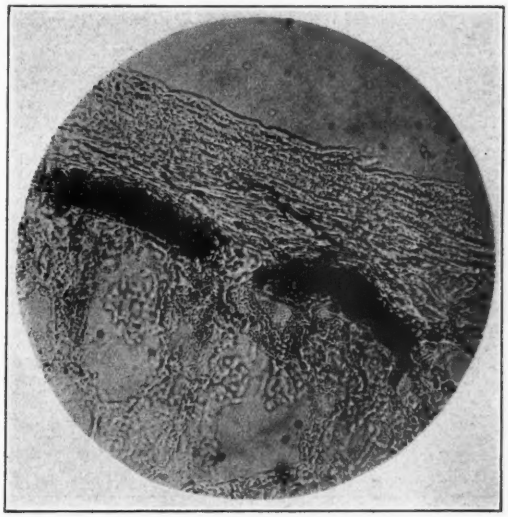

Fig. 2.—This illustration is an unstained microphotograph of a thick frozen section (20 microns), magnification approximately ×800. The tortuous vessel seen running obliquely toward the alveolar tissue is an injected bronchial artery. The two larger vessels immediately beneath the pleura are pulmonary veins filled with carmine red. The pleural vessel, as shown, looks as though it enters the larger pulmonary vein, but this is not true.

retained on the arterial side. If any anastomosis exists between the bronchial artery and pulmonary artery, black pigment should have been found in the vessels containing red and green colors. On the other hand many small vessels as well as larger vessels contained red and black colors. This shows definitely the anastomosis of the bronchial arteries with the pulmonary veins.

In the experiments cited above, it occurred to us that anthracitic pigment in the subpleural and perivascular lymphatics might have been in some instances mistaken for injected blood vessels. It also seemed advisable to carry out some experiments with the aorta unclamped, thus preventing back pressure on the pulmonary veins.

Thus, injections were made in five dogs using carmine red in the bronchial circulation and a mixture of zinc chromate in glycerin and egg albumen in the pulmonary artery. The aorta was not clamped.

The right pulmonary artery was exposed and a cannula was placed in the left carotid artery. A ligature was passed around the right pulmonary artery, but this ligature was not tied. A long needle attached to a syringe was inserted into the right pulmonary artery. A simultaneous injection of the zinc chromate into the right pulmonary artery and carmine red into the carotid artery was made, while traction was exerted against the right pulmonary artery with the ligature mentioned

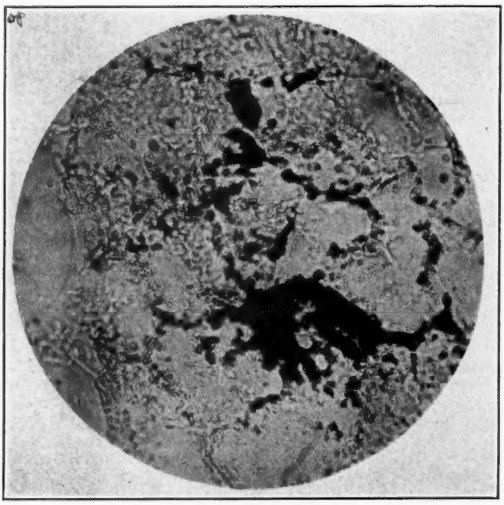

Fig. 3.—Unstained microphotograph of a frozen section of right lung injected with zinc chromate (20 microns), magnification approximately ×800. The photographer made a double exposure to show the injected pulmonary arterioles as well as the injected alveolar capillaries derived from the pulmonary arterioles. Examination of many similar areas did not show carmine red mixed with the zinc chromate in these capillaries.

above. Traction made by pulling upon the ligature prevented blood from passing into the right lung after the injection of zinc chromate had been made. The zinc chromate was injected at a pressure of approximately 30 millimeters of mercury. The bronchials, had been injected retrograde through the carotid artery. The right pulmonary artery and the trachea were clamped almost simultaneously with the injection of the two systems. Formalin solution was injected into the trachea to fix the soft tissue before collapse. The tracheal tree was filled with formalin without overdistention.

Nothing could pass into the right lung after the beginning of the injection, excepting by the way of the bronchial vessels, because the

right pulmonary artery had been occluded. The left lung was used as a control.

The results obtained in the last group of experiments were the same as the results reviewed in the first thirty-five experiments. We were fortunate to receive an injection of the dye in the pulmonary artery which went down into the alveolar capillaries without finding any of the dye in the pulmonary vein capillaries. We did find, however, the carmine red which had been injected into the bronchials, in small capillaries which could be traced to pulmonary veins. The larger pulmonary veins showed carmine red in all the vessels. Zinc chromate could not be found in any of the vessels of the left lung.

A study of the literature shows relatively few references to the vasa vasorum supplying the walls of the pulmonary arteries and veins. Cohnheim[21] mentioned these vessels in 1889. In these experiments the walls of the pulmonary artery show a heavy injection with the black pigment and we conclude, therefore, that these vessels receive arterial blood by way of vessels which arise in the ascending aorta, probably in part at least from the bronchials. The pulmonary veins, excepting the larger branches, show fewer injected vasa vasorum.

## SUMMARY

1. We have confirmed in the living animal without changes in the intrathoracic pressures, the work of William Snow Miller, i. e., there is no anastomosis between the bronchial and pulmonary arteries and that the bronchial arteries anastomose with the pulmonary veins.

2. There is a gross anastomosis between the intercostal, internal mammary, esophageal and bronchial arteries in the dog.

3. The visceral pleura in the dog receives blood from the bronchial arteries or from vessels anastomosing with them.

4. The vasa vasorum of the pulmonary artery and vein are vessels which are probably derived, in part at least, from the bronchial arteries.

The viridine green (Ansbacher-Siegle) used in these experiments was supplied by the Henry Seim Company. The dye is composed of the following ingredients: 74 per cent aluminum hydroxide, 8.7 per cent sulphate of tetraethyldiaminotriphenyl carbinol anhydride, 17.3 per cent chloromethyl-derivative of dimethyl dihydro-p-toluidine.

## REFERENCES

1. Virchow, R.: Ueber die Verstopfung der Lungerarterie. Notizen Gebiete Natur, u. Heilk. **37**: 26-31, 1846. Gesamelte abhandlunger. Frankfurt 1856, 295, Experiment XX.
2. Küttner: Beitrag zu den Kreislaufsverhaltnissen der Froschlunge. Virchow's Arch. f. Path. Anat. **61**: 21, 1874. Beitrag zur Kenntniss der Kreislaufsverhaltissen der Saugethierlunge, ibid. **73**: 476, 1878.
3. Cohnheim, J.: Vorlesungen über allgemeine Pathologie, Berlin, 1882, p. 207.
4. Litten, M.: Untersuchungen uber den haemarrhogischen Infarkt, und uber die Einwirkung Arterieller anaemie auf das lebeude Gwebe. Ztschr. f. klin. Med. **1**: 131, 1880.

5. Konigstein, H.: Zur morphologie und Physiologie des Gefass systems am Respirationstrakt. Anat. Hefte. **22**: 309, 1903.
6. Tigerstedt, R.: Der Kleine Kreislauf, Ergebu. d. Physiolog. 1903, II Pt. 2, 528.
7. Zuckerkandl, E.: Ueber die anastomosen der vena pulmona les mit den Bronchialvenen. Sitzungsber. d. k. akad. d. Wissensch., math, natur. Kl., **84**: pt. 3, 310, 1881. Ueber Verbindungen Zwischen den arteriellen Gefassen der menschlichen Lungen. idem, **87**: pt. 3, 171, 1883.
8. Ghoreyeb, A. A., and Karsner, H. T.: A Study of the Relation of Pulmonary and Bronchial Circulation, J. Exper. med. **18**: 500-506, 1913.
9. Karsner, H. T., and Ash, J. E.: Studies in Infarction: Experimental Bland Infarction of the Lung, J. M. Research **27**: 205, 1912.
10. Coryllos, Pol N., and Birnbaum, G. L.: Circulation in Compressed Atelectatic and Pneumonic Lung. Arch. Surg. **19**: 1346-1425, 1929.
11. Miller, W. S.: The Structure of the Lung, J. Morphology, **8**: 165-181, 1893.
12. Miller, W. S.: Anatomy of the Lungs, Reference Handbook of the Medical Sciences, Wm. Wood & Co. vol. V, 575-586.
13. Miller, W. S.: Das Lungenlappchen, Seine Blut-und Lymphgefasse, Arch. f. Anat. u. Physiol. anat. abt. p. 197, 1900.
14. Miller, W. S.: The Arrangement of the Bronchial Blood Vessels, Anat. anz. **27**: 432, 1906.
15. Miller, W. S.: The Vascular Supply of the Pleura Pulmonalis, Am. J. Anat. **7**: 389, 1907.
16. Miller, W. S.: The Lobule of the Lung and Its Blood Vessels, J. Morph. **24**: 459, 1913.
17. Haller, A.: Icones Anatomicae. Tabulas Arterium Bronchialium, Gottingen, B. A. Vandenhoecku, 1756, Pt. 8, p. 31.
18. Hyrtl, J.: Die Carroscions-Anatomie und ihre Ergebnisse, Vienna, Wilhelm Braumullèr, 1873, p. 93.
19. Henle, F. G. J.: Handbuch der Eigenweidslehre des Menschen Braunschweig, F. Viewig u. Sohn, 1866.
20. Luschka, H.: Die Anatomie des Menschen. Tubingen, H. Laupp. **2**: 316, 1863.
21. Cohnheim, J.: Lecture on General Pathology, **1**: 218, 1889.

# METASTATIC CARCINOMA OF THE HEART SECONDARY TO PRIMARY CARCINOMA OF THE LUNG*†

CHARLES H. MEAD, M.D.
MINNEAPOLIS, MINN.

EXAMINATION of the literature regarding primary or secondary carcinoma of the heart shows the condition to be very rare. Conclusions regarding frequency of occurrence of carcinoma of the heart are unsound when based upon reports of tumors by early writers, for there is some difference of opinion as to whether they were real mesoblastomas or organized thrombi which had undergone metamorphosis. Autopsy statistics, however, based upon 10 large series of cases, establish the infrequency of occurrence of carcinoma of the heart in relation to other lethal diseases. Primary carcinoma of the heart occurred in 0.03 per cent of autopsies, and metastatic carcinoma of the heart occurred in 0.5 per cent of autopsies. Metastatic sarcoma of the heart occurred in 0.01 per cent of autopsies. The occurrence of metastatic cardiac deposits secondary to carcinoma in general is much greater than found in general autopsy statistics. Blumensohn reviewed 1,078 cases of carcinoma in general and found metastatic cardiac deposits in 34, or 3.1 per cent of cases. He found 12 cases, or 7.5 per cent, with metastases to the heart in a series of 160 cases of sarcoma. The frequent involvement of the heart was probably present because secondary sarcomatous growths are most common in the lungs. Bodenheimer concluded from the literature that 7.5 per cent of cases of carcinoma with metastases involve the heart. It is because of the infrequency of occurrence of the condition that this case is reported.

## CASE REPORT

White male, sixty-five years of age, admitted to the University of Minnesota Hospital on October 1, 1930. Death occurred eleven days later.

*History.*—The patient had enjoyed good health until the latter part of June, 1930. He developed intermittent, dull, aching pain in the right chest at this time. The pain was of variable intensity and persisted for a period of two weeks. He developed a slight cough, occasionally associated with blood-streaked sputum, shortly after the onset of the pain. He was not confined to his home or bed and was treated by his family physician.

The patient began to lose weight and became easily fatigued coincident with the onset of his complaints. Approximately one month after the onset of symptoms he became free from cough and sputum and felt perfectly normal except for marked fatigue following casual exertion. A chill, associated with a rise of temperature, occurred on September 1, 1930. He recovered in a few days but continued thereafter to have intermittent chills associated with elevations of body temperature.

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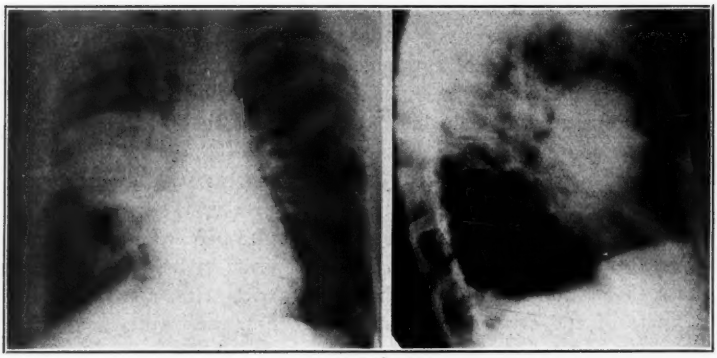
*From the Department of Surgery and the Cancer Institute of the University of Minnesota, Minneapolis, Minn.
†Received for publication, December 3, 1931.

Past history, family history and marital history revealed nothing of importance. Course before admission to the hospital: Careful notes were kept by the attending physician, Major F. Sedgely of the U. S. Veterans' Hospital, Fort Snelling, Minnesota, before the patient was referred to the University Hospital. Blood examination at the time of onset of the chills and fever showed a white blood cell count of 31,000 per cubic millimeter, 92 per cent of which were neutrophilic polymorphonuclear leucocytes. The blood was found to be negative for typhoid, paratyphoid and malaria. The urine was normal. All tuberculosis tests were negative. Physical examination revealed only occasional râles in the right chest.

The patient was confined to bed on October 1, 1930. Electrocardiograms, blood examinations and urinalyses were made at this time, all of which failed to give any additional information. Weight records showed a thirty-four pound loss.

Intermittent coarse râles in the right anterior thorax were the only abnormal findings noted on repeated physical examinations. The patient's general condition remained unchanged from the time of his confinement to bed until he was admitted to the University Hospital. On admission he complained chiefly of general malaise and intermittent chills and fever, all other symptoms having been absent for the preceding month.



*A* *B*

Fig. 1.—*A*, An anteroposterior roentgenogram demonstrating an annular mass with well defined contours in the right chest. The presence of fluid in considerable quantity in the right pleural space can be made out. The left costophrenic angle is seen to be obliterated, suggesting pleural adhesions, and there is no evidence of fluid in this pleural space.

*B*, A lateral roentgenogram showing the mass in the right chest to be located more anteriorly than posteriorly and definitely anterior to the mediastinum.

Physical examination at the time of admission to the hospital: The patient's general condition was good except that there was evidence of loss of body weight. A dull percussion note and diminished breath and voice sounds over the right anterior thorax were found on examination of the chest. Neither râles nor rubs were detected. The heart was considered to be entirely normal. Examination of the prostate gland showed it to be slightly enlarged but otherwise normal. The remaining physical examination revealed no origin for a possible metastatic tumor of the lung. Laboratory data at time of admission to the hospital: Blood—hemoglobin 70 per cent, red blood cells 3,160,000, white blood cells 19,920; differential white blood cell count—neutrophilic polymorphonuclear 74, eosinophilic polymorphonuclear 4, lymphocytes 19, monocytes 3. Wassermann test was negative. Urine—specific gravity 1020. Acid reaction. Neither sugar nor albumin. Microscopic—an occasional granular cast, red blood cell and white blood cell.

*Diagnostic Procedures.*—Examination by x-ray (Figs. 1 *A* and *B*) on October 16, 1930, demonstrated an annular mass with well-defined contours in the right chest in its midportion near the hilum of the lung. A lateral plate showed the mass to be almost spherical and in the anterior mediastinum. A thickened interlobar fissure could be made out running anterior and posterior to the mass. There was also a distinct thickening of the pleura which was seen with the patient in the upright position. In the right lateral decubitus the presence of free fluid in the pleural space could be made out. The left costophrenic angle was obliterated, its appearance suggesting pleural adhesions. There was no evidence of fluid in the left pleural cavity. There was no definite evidence of other parenchymal pathologic condition in the lungs. From the x-ray examination, the appearance of the mass in the right chest suggested an encapsulated interlobar effusion, tumor of the pleura, carcinoma of the lung or possibly some type of cyst such as a dermoid or echinococcus cyst. An encapsulated interlobar effusion was considered the most probable diagnosis.

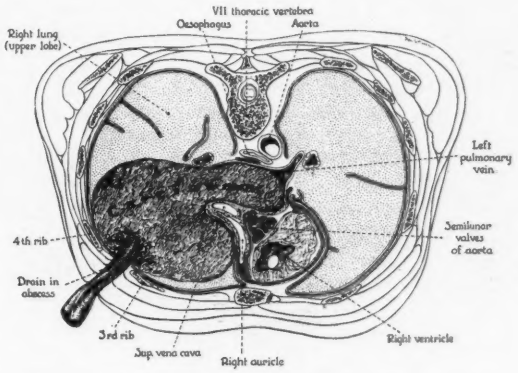



Fig. 2.—A photograph of a sagittal section through the sternum, lung and heart. A firm neoplastic mass is seen within the lung substance. The mass is encroaching upon the sternum anteriorly and has extended through the right pulmonary vein laterally into the left atrium of the heart. It appears to fill the left atrium, and it is seen to extend through the bicuspid valves into the left ventricle.

Thoracentesis of the right chest was performed with the patient in the lateral decubitus position on an orthopedic table, such that puncture was easily made from beneath the table. Twenty-five cubic centimeters of clear, amber colored fluid were aspirated with difficulty. Examination of the fluid showed the presence of albumin. Microscopic examination showed red blood cells but no tumor cells and no bacteria. Bacteriologic examination showed no growth upon agar or broth.

Bronchoscopy was performed by Dr. K. Phelps, and all of the main bronchi of the right lung were examined. The orifice of the middle lobe bronchus was open, and no obstruction was seen as far as it was possible to visualize the lumen. No pus, blood, or other discharge was observed. The other bronchial orifices were also open. No bronchial tumor was seen.[50]

*Diagnosis.*—Although some of the clinical symptoms were suggestive of a neoplasm of the lung, a definite diagnosis of this condition could not be made. Before the patient's admission to the hospital he had had recurrent attacks of chills and fever. After a few days in the hospital he felt much better but continued to have an occasional chill with a slightly hectic type of temperature curve varying from normal to 101 degrees Fahrenheit. There had been no sputum except in June, at the onset of the trouble, at which time a little rusty sputum had been brought up. The oc-

currence of these symptoms during the course of the patient's illness strongly suggested an infectious pulmonary lesion. Interpretation of the x-ray films favored a diagnosis of an encapsulated pleural effusion. With these considerations in mind a preoperative differential diagnosis of an interlobar empyema, dermoid cyst of the lung with associated infection, or malignant tumor of the lung was made.

*Therapeutic Procedures.*—Because of the infectious features it was thought best to proceed as though the lesion was inflammatory in character. On October 20, 1931, operation was performed by Dr. O. H. Wangensteen. Under procaine infiltration anesthesia an incision was made in an oblique direction extending from the right lateral margin of the sternum at the level of the second rib to the level of the sixth rib in the anterior axillary line. The pectoralis major muscle was split in the direction of its fibers, and the anterior portions of the third, fourth and fifth ribs were resected. This incision and opening allowed direct access to the pleura immediately

Fig. 3.—A diagrammatic transverse section through the carcinoma of the lung and heart at the level of the seventh thoracic vertebra.

overlying the pulmonary mass. The visceral pleura moved freely beneath the parietal pleura, although the mass could be easily felt within the lung.

It was felt best to treat the condition as inflammatory in nature, and a gauze pack was applied over the parietal pleura to stimulate adherence of the visceral pleura to the chest wall before drainage of the suspected pulmonary lesion was undertaken.

The pulmonary mass was drained on October 27, 1930, under local anesthesia. The old incision was reopened, the pectoralis major muscle split again at the site of previous suture and the gauze pack removed. A needle was introduced into the lung at the site of the lesion and pus was aspirated. A small incision in the lesion was made, and 5 to 8 c.c. of pus were obtained. The tissue comprising the pulmonary mass was yellowish red in color and resembled granulation tissue. A small tube and gauze pack were introduced into the incision in the pulmonary mass and the skin was closed with a few stitches.

The tissue from the lesion in the lung was examined microscopically, and it proved to be definite bronchogenic carcinoma. The patient became irrational immediately

following this procedure, although he had been mentally obtunded for a few days previously. Otherwise his general condition was essentially unchanged for about fifteen hours. He then suddenly developed dyspnea, became very weak and cyanotic, the pulse became very rapid, and death occurred within the first twenty-four hours following operation. The manner of death gave the impression of shock without apparent cause, as no evidence of bleeding was found.

*Autopsy Findings.*—The body was 167 cm. in crown-heel length and weighed approximately 140 pounds. It presented a very pale appearance. The bodily development was good but there was evidence of marked loss of weight. Further examination revealed an unhealed, oblique incision 13 cm. long in the right anterior thoracic region. The lower portion of this incision was open for drainage. The scar of an old healed incision was present in the region of the lower right rectus abdominis muscle.

The peritoneal cavity presented scattered adhesions of the omentum to the anterior parietal peritoneum. The appendix was absent and there were old pericecal

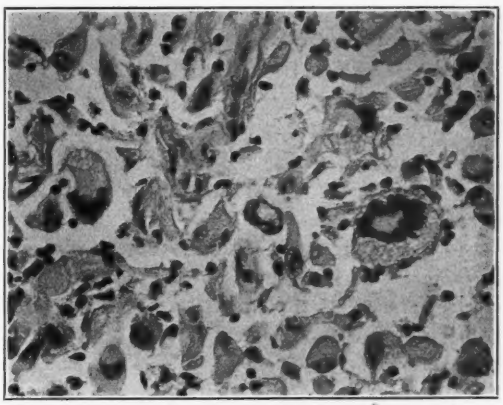


Fig. 4.—A high power lens view of typical bronchogenic carcinomatous tissue taken from the pulmonary mass (× 350).

adhesions. There were approximately 1000 c.c. of clear fluid present in each pleural space. There were pleural adhesions about the right lung apex and in the region of the anterolateral wall of the right pleural cavity. The latter were very extensive as they were about the site of rib resection and pulmonary drainage. There were also a few scattered adhesions in the left pleural cavity.

An area of 4 cm. in diameter, consisting of both the overlying pericardium and the myocardium of the right atrium, was invaded by carcinoma tissue. A mass of carcinoma tissue, originating from the right lung had entered and grown within the right pulmonary vein to the left atrium, Figs. 2 and 3. The mass had enlarged greatly within the atrium causing a marked dilatation of the atrial wall. The carcinoma tissue had also grown through the bicuspid valves and projected into the left ventricle. The carcinomatous mass in the left atrium so completely filled the cavity that it was difficult to understand how blood could pass from the pulmonary veins into the heart, Fig. 2. The tip of the process which extended into the left ventricle was granular in appearance and suggested the possibility of carcinomatous emboli from this source. There was no invasion of the endocardium at any point.

The right lung weighed 480 grams and was partially collapsed. A large area of the contiguous portions of the upper and middle lobes was composed of carcinomatous

tissue. The entire mass measured 12 × 8 cm., and was adherent to the anterior thoracic wall at the site of previous rib resection. The tumor tissue was a yellow-brown color and firm in consistency throughout, except in its anterolateral portion where an area 4 × 4 cm. was very necrotic and had the gross appearance of an abscess, Fig. 4. The origin of the tumor was apparently from a small bronchus in the upper portion of the middle lobe with extension into the lower portion of the middle lobe and also through the interlobar pleura into the lower portion of the upper lobe. Extension of the mass had taken place in an anterolateral direction and toward the right lung hilus, the greater portion of the mass lying in the anterior portion of the lung substance. The abscessed portion of the tumor had been drained surgically. The bronchial lymph nodes were extensively invaded.

The left lung weighed 780 grams and presented evidence of broncho-pneumonia but no evidence of carcinoma. The spleen weighed 275 grams and presented in its lower pole an infarct measuring 2.5 cm. in diameter, which was grossly an embolic metastatic carcinomatous process. The infarct was apparently old as it was white in color and its margins were somewhat indistinct.

*Microscopic Findings*: Examination of the tissue removed from the pulmonary neoplasm at autopsy showed it to be a typical bronchogenic carcinoma, Fig. 4. Sections of the splenic infarct showed this structure to be composed of typical bronchogenic carcinoma tissue with almost complete exclusion of splenic parenchyma.

## COMMENT

Tedeschi states that Boneti was the first person to call attention to tumor of the heart but that Albers in 1835 presented the first authentic report of primary cardiac tumor.

*Pathology.*—Fibromas, myxomas, fibromyxomas and rhabdomyomas are the most common primary tumors of the heart; lipomas, angiomas and teratomas are rare.[8, 27, 19, 34] Until 1927 approximately 150 primary tumors of the heart had been reported[7, 18, 22, 30, 34, 39, 40] and about 40 of these cases were sarcoma.

Metastases to the heart are more common than primary cardiac neoplasms,[20] and of these metastatic tumors carcinomas are more common than sarcomas in general autopsy statistics.[34] The condition when present is usually associated with widespread carcinoma of other organs, the lungs being the most commonly involved.

A primary tumor of the lung was first described by Bayle in 1810. More detailed information dates from the studies of Wolf (1895) and Passler (1896). The present literature is so extensive that in 1911 Adler was able to collect 374 cases of carcinoma and 90 of sarcoma of the lung. Since 1922 approximately 120 new cases have been reported. Primary malignant tumors of the lung form about 1 per cent of all cancers and occur most commonly in males. They are most frequently located in the right lung. These tumors are usually classified as arising from the bronchial lining epithelium, bronchial mucous glands or pulmonary alveoli. Metastases are numerous and widespread, the vascularity of the organ and frequent invasion of blood vessels resulting in marked dissemination of the tumor cells. The most frequent sites of metastases are the bronchial lymph nodes and the liver.

Carcinomatous metastases to the heart may be derived from practically any organ of the body in which malignant disease is found. Cardiac involvement has been reported from carcinoma when the primary lesion was in the trachea,[29] tongue,[31] submaxillary gland,[50] skin of face,[37] esophagus,[33, 46] stomach,[33] pancreas,[4] breast,[18, 22] uterus,[43] testes,[25] and lung.[1, 19, 47] Cardiac involvement has been reported from sarcoma when the primary lesion was in the kidney,[5, 37, 42] skin,[16] lymph glands,[2] bone,[22] eye,[16] nasal passages,[53] sternum,[49] and ilium.[3] A pigmented soft mole of the labium majus[16] has been reported to have been the source of cardiac metastases. Associated pulmonary metastases were found in almost all reported cases of metastatic carcinoma of the heart. Pic and Bret (1891) stress this point and state that the lungs are always affected simultaneously with the heart. Tumor cell emboli are very frequent within the lungs, the cells being arrested in small arterioles and venules. The emboli, however, are much more frequent in the lungs than are the secondary tumors of these organs. The relative frequency of lung metastases, compared with the relative infrequency of secondary cardiac involvement, suggests that the lungs and adjacent tissues arrest and support cancerous growth more effectively than cardiac tissue. Possibly the rapid cardiac vascular flow may be related to this relative infrequency of metastases to the heart.[12]

Adler[1] (1911) found 39 metastases to the pericardium in an analysis of 374 cases of primary carcinoma of the lung, and of these cases 30 involved the heart proper. One case had perforated but not invaded a pulmonary vein, and one had grown along a pulmonary vein into the left atrium. None had penetrated the pulmonary vessels among 90 cases of sarcoma. Examination of 139 cases of carcinoma of the lung,[47] reported at the London Hospital from 1907 to 1925, revealed pericardial invasion in 62 cases and "frequent" direct invasion of pulmonary vessels, but apparently no infiltration of the cardiac chambers. The type of metastasis to the heart from carcinoma of the lung described in this paper would occur in 0.26 per cent of cases of primary carcinoma of the lung, on a basis of Adler's figures and in a still smaller percentage of cases of carcinoma of other tissues with metastases.

A small number of metastases to the heart are believed to occur by lymphatic conduction.[34] The tracheobronchial glands drain pleura, lungs, myocardium and pericardium and may conduct invasion in a retrograde fashion from tumors of the lungs and pleura.[2, 53] Direct extension to the heart through growth of strands of tumor cells into the surrounding tissue interstices may occur. The growth of these strands is usually continuous and guided by dense fascia or other resisting tissue. Direct extension is considered possible from tumors which are primary in the lung or mediastinum.[26, 40] It is stated,[34] however, that the heart is seldom involved by continuity or contiguity from neighboring

organs. A survey of a few large series of cases of primary carcinomas of the lung seems to substantiate this view.

Metastases to the heart muscle are usually described as being blood borne. Only a limited number of cases of neoplastic heart thrombosis which offer proof of blood borne invasion are reported.[2, 25, 51] Tumor cells in the circulating blood and small bits of tumor in blood clots, carcinoma cells in the center of mural and valvular cardiac thrombi,[49] and strands of tumor cells in the lumen of coronary vessels without evidence of neighboring metastatic nodules[47] have been described as further evidence of favoring blood borne metastases. Implantation grafts in the endocardium are probably a result of neoplastic emboli entering the blood stream through the lymphatic duct, bronchial or azygos veins and then through the superior vena cava to the right atrium and ventricle.[34] Some writers feel that the right and left muscular heart walls are involved in equal ratio,[10] while others[15, 35] feel that the right heart is more frequently attacked, especially the right ventricle. Primary tumors apparently involve the right atrium most frequently. Secondary tumors are frequently found as small white, shotty accumulations of cell growth, widely scattered throughout the muscular wall and in close proximity to the vascular supply.[51] Vegetation-like masses may encroach upon a heart chamber or valve.[25, 43] Pedunculate tumors may be free in the heart cavities.[51] Massive atrial infiltration may produce an anemia of the arteries, due to encroachment on the heart cavity.[38] Tumors involving the musculature may disturb the cardiac mechanism by changes in the cardiac muscle fibers[51] or cause anginal attacks by occluding coronary vessels.[24] The conduction mechanism[32] may be altered causing bradycardia with dissociation.[4]

Pedunculated grafts or free tissue masses in a heart cavity may produce multiple emboli, valvular insufficiency or stenosis.[43] It is interesting to compare the embolic possibilities of this process with the peripheral embolic processes originating from suppurative conditions within the lung. Eagleton[17] believed that approximately 12 per cent of metastatic abscesses were of pulmonary origin. Shorstein[45] in a study of 63 cases of pulmonary suppuration found 20 per cent to have brain abscesses. Eagleton[17] has shown that pulmonary suppurative metastases to the brain involve the left cerebrum three times more frequently than the right. Eagleton, Shorstein and Gardner conclude that the most probable route of metastases from pulmonary suppurative processes is via the pulmonary veins through the left atrium, left ventricle and aorta. It is commonly believed that the pulmonary veins act as a tumor cell conveyance for carcinoma of the lungs, pleura or mediastinal lymph nodes. The tumor cells are described as circulating through the left atrium, left ventricle and coronary circulation of the heart muscle.[47] It has also been considered possible for emboli to pass by this general

route into the peripheral circulation. The manner of growth of the above described metastatic carcinomatous thrombus demonstrates conclusively the accuracy of this theorem.

*Symptoms.*—So-called cardinal symptoms of carcinoma of the heart emphasized by some writers,[13, 14, 23] are cyanosis and precordial pain, associated with frequent recurrent hemorrhagic pericardial effusion. The hemorrhagic pericardial effusion indicates pericardial tumor transplant as a rule, if there are other metastases and the pericardial fluid contains cancer cells. It seems probable in the case here reported that the intracardiac obstructive process was very gradual and that cardiac insufficiency became predominant only when unusual activity was required of the circulatory system. Thus anemia due to almost complete obstruction of the left atrium was responsible for the symptoms of shock and the sudden death.

The usual symptoms of carcinoma of the heart are dyspnea, profuse perspiration, restlessness, nausea, repeated emesis, dizziness, weakness, cough and thirst. Almost invariably the symptoms are obscured by the primary disease picture. When they are observed they are generally attributed to the primary neoplastic condition and neglected. It is regrettable, from a diagnostic point of view, but nevertheless true that such cases presenting an inevitable lethal outcome are usually followed in a rather desultory fashion. Consequently the recognition of symptoms of cardiac involvement is not recorded, as the findings are attributed to the primary disease.

*Diagnosis.*—Roentgenography and fluoroscopy are probably the best diagnostic aids available since changes in cardiac contour with a large tumor extending in one direction only, and failing to simulate the usual cardiac morbid states, might be diagnostic in conjunction with the other physical findings. In general, however, there are no pathognomonic signs of carcinoma of the heart, roentgenographic aid is not decisive, and there are no laboratory findings, other than microscopic examination of pericardial effusions, that will assist in the diagnosis. The consensus of opinion seems to be that the clinical diagnosis of carcinoma of the heart is rarely if ever made. The symptoms may be absent, mild or severe. Usually the symptomatology is little different from ordinary myocardial insufficiency or the general failure noted in the toxemia of widespread malignant disease.

Keller and Callender report the successful operative removal of neurofibroma arising from the pericardial pleura. The authors concluded from their review of the literature concerning tumors in this region that it was not possible to state that no other reports of operative removal existed. They considered the procedure, however, as one of rare occurrence, and they had found no definite evidence of a previous similar operative procedure. In a discussion of this paper Dr. H. Lilienthal

stated that he had performed a similar operative procedure in December, 1929.

Suffice it to say that the surgical removal of tumors of the pericardium has been successful, but only very rarely attempted, so that no standardized procedures in these cases have been developed. No operative methods have been devised for the treatment of neoplasms of the epicardium, myocardium or endocardium.

## SUMMARY

1. Primary or secondary carcinoma of the heart is very rare.

2. Metastasis to the heart is more common than primary cardiac neoplasm.

3. Carcinomatous metastases to the heart may occur from practically all organs of the body in which malignant diseases are found.

4. Carcinoma of the lungs is the most common source of carcinoma of the heart.

5. Metastatic carcinoma of the heart is almost always associated with pulmonary metastases.

6. In all probability metastases to the heart are blood borne.

7. Peripheral metastases from carcinoma of the lungs, in all probability, pass most frequently through the pulmonary veins, left atrium, left ventricle and aorta.

8. Metastatic carcinomatous thrombosis of the heart with resulting carcinomatous embolic phenomena is very rare.

9. There are no pathognomonic signs of carcinoma of the heart.

10. Clinical diagnosis of carcinoma of the heart is rarely, if ever, made.

## REFERENCES

1. Adler, I.: Primary Malignant Growth of the Lungs and Bronchi, New York, 1912, Longmans, Green & Co.
2. Aubertin, C.: Sur un Cas de Thrombose Neoplasique du Coeur droit, Arch. de Med. Exper. et d'Anat. Path. **17**: 197, 1905.
3. Bardenheuer, F. H.: Zur Kenntnis der Metastasierung Bösartiger Geschwülste in Herzen, Centralbl. f. allg. Path. u. path. Anat. **34**: 337, 1924.
4. Barthelemy: Note sur deux Cas de Cancer secondaire du Coeur, Bull. Soc. Anat. de Paris **54**: 551, 1879.
5. Batzdorff, E.: Ein Herz mit Hypernephrommetastasen, Berl. klin. Wchnschr. **50**: 1504, 1913.
6. Bayle: Rech. sur le Phthisie Pulmon., 1810, Paris (quoted by Ewing).
7. Beck, C. S., and Thatcher, H. S.: Spindle Cell Sarcoma of the Heart, Arch. Int. Med. **34**: 831, 1925.
8. Berthensen, L.: Aus frage von der Diagnose Primärer Neoplasmen des Herzens Myxom des Linken Vorhofes, Virchow's Arch. **132**: 390, 1893.
9. Blumensohn: Inaugural dissertation, Basel, 1907 (quoted by Peters and Milne).
10. Bodenheimer: Beitr. z. Path. der Krebsartigen Neubild. in Herzen, Inaugural dissertation, Bern, 1865 (quoted by Berthensen, Morris, Tedeschi, et al.).
11. Boneti, Theophili: Sephulchreturer sur anatomia practica ex cadaveribus morbe denatis, etc., Miscellanea, Geneva, 1700, J. III, p. 505 (quoted by Tedeschi).
12. Burrows, M. T.: The Mechanism of Cancer Metastasis, Arch. Int. Med. **37**: 453, 1926.

13. Cashion, W. A.: Metastatic Carcinoma of the Heart, U. S. Vet. Bur. M. Bull. **3**: 810, 1927.
14. Coffin, T. H.: Carcinoma of the Heart, The Post-Grad. M. J. **24**: 41, 1909.
15. Cornil: Thése de Paris, 1903 (quoted by Peters and Milne).
16. Delherm and Laignel-Lavastine: Un Cas de Sarcome Melanique (Sarcome Secondaire du Coeur), Bull. Soc. Anat. de Paris **78**: 125, 1903.
17. Eagleton, W. P.: Brain Abscess, New York, 1922, The Macmillan Co.
18. Ehrenberg, L.: Zwei Fälle von Tumor in Herzen; ein Beitrag zur Kenntnis der Pathologie und Symptomatologie der Herztumoren, Deutsches Arch. f. klin. Med. **103**: 293, 1911.
19. Ewing, James: Neoplastic Diseases, Philadelphia and London, 1928, W. B. Saunders Co.
20. Ferel, F. C.: Two Cases of Primary Neoplasm of the Cardiac Valves, Internat. Clinic **4**: 147, 1919.
21. Gardner, W. J.: Metastatic Abscesses of the Brain of Pulmonary Origin, Report of 2 Cases, Arch. Neurol. & Psychiat. **19**: 904, 1928.
22. Goldstein, H. I.: Tumors of the Heart, New York M. J. **115**: 97, 1922.
23. Hirschfelder, A. D.: Diseases of the Heart and Aorta, Philadelphia and London, 1918, J. B. Lippincott Co.
24. Ingram, T. D.: Cancerous Heart (?) With Dilated Right Ventricle; Sudden Death With Symptoms of Angina Pectoris, Tr. Path. Soc. Philadelphia, **8**: 59, 1877.
25. Kasthack, A. A.: Case of Carcinoma of the Testis in a Young Man With Metastatic Deposits Lying Free in the Heart and Inferior Vena Cava, J. Path. & Bact. **5**: 78, 1898.
26. Kapsinow, R.: Cancer of the Heart, Ann. Surg. **63**: 161, 1926.
27. Karrenstein: Ein Fall von Fibroclastimyxom des Herzens und Kasuistisches zur Frage des Herzgeschwülste, besonders der Myxome, Virchow's Arch. f. path. Anat. **194**: 127, 1908.
28. Keller, W. I., and Callender, G. R.: Neurofibroma Arising on the Pericardial Pleura, Ann. Surg. **92**: 666, 1930.
29. Linell, E. A.: An Unusual Cause of Death From Cancer, Brit. M. J. **1**: 872, 1922.
30. Link, R.: Die Klinik der Primären Neubildungen des Herzens, Ztschr. f. klin. Med. **67**: 292, 1909.
31. Lina, J. R.: Three Cases of Metastatic Neoplasms of the Heart, New York Path. Soc. **24**: 78, 1923-24.
32. Martin, C. F., and Klotz, Oskar: Extensive Sarcoma of the Heart Involving the Bundle of His, Am. J. M. Sc. **140**: 216, 1910.
33. Moore, N.: Cases of Carcinoma of the Heart, Tr. Path. Soc. London, **37**: 172, 1886.
34. Morris, L. M.: Metastases to the Heart From Malignant Tumors, Am Heart J. **3**: 219, 1927-28.
35. Napp, O.: Ueber Sekundäre Herzgeschwülste, Ztschr. f. Krebsforsch. **3**: 282, 1905 (quoted by Morris).
36. Passler, Hans: Ueber das primäre Carcinom der Lunge, Virchow's Arch. f. path. Anat. **145**: 191, 1896.
37. Peterson, R. P.: The Relationship Between the Clinical and Pathological Findings in Primary Pulmonary Malignancy, Canadian M. A. J. **22**: 333, 1930.
38. Pavlowsky, R.: Beitrag zum Studien der Symptomatologie der Neubildungen des Herzens, Berl. klin. Wchnschr. **32**: 393, 1895.
39. Perlstein, I.: Sarcoma of the Heart, Am. J. M. Sc. **156**: 214, 1918.
40. Peters, H. LeB., and Milne, L. S.: Secondary Tumors of the Heart, N. Y. M. J. **94**: 383, 1911.
41. Pic, A., and Bret, J.: Contribution a L'Etude du Cancer Secondaire du Coeur, Rev. de Med. **2**: 1022, 1891.
42. Rabe and Morel: Sarcome du Coeur Secondaire a un Sarcome du Rein, Bull. Soc. Anat. de Paris **76**: 400, 1901.
43. Rist, E., and Rolland, J.: Un Cas D'Endocardite Vegetante Epitheliomatouse Secondaire avec Embolics Pulmonaires, Ann. de méd. **13**: 538, 1923.
44. Rosahn, P. D.: The Incidence of Primary Carcinoma of the Lung, Am. J. M. Sc. **179**: 803, 1930.
45. Shorstein, G. I.: The Shorstein Lecture on Abscess of the Brain in Association With Pulmonary Disease, Lancet **177**: 842, 1909.

46. Siegal-Delval and Marie: Cancer Secondaire du Coeur, Bull. Soc. Anat. de Paris **80**: 702, 1905.
47. Simpson, S. L.: Primary Carcinoma of the Lung, Quart. J. Med. **22**: 413, 1929.
48. Taub, S. J.: Primary Bronchogenic Carcinoma of the Lung, M. Clin. N. Am. **12**: 279, 1928.
49. Tedeschi, A.: Beitrag zum Studien der Herzgeschwülste, Prager med. Wchnschr. **18**: 121, 1898.
50. Vinson, P. P., Moersch, H. J., and Kirklin, B. P.: Value of Bronchoscopy in Diagnosis of Malignant Conditions of the Lungs, J. A. M. A. **91**: 1439, 1928.
51. Warthin, A. S.: A Primary Polymorphous-Cell Sarcoma of the Nose, N. Y. M. J. **69**: 879, 1899.
52. Wolf, K.: Fortschr. d. Med. 1895 (quoted by Ewing).
53. Wolf and Geit: Lymphosarcoma Mediastinal, Envahissement du myocarde, Bull. Soc. Anat. de Paris **92**: 340, 1922.

# DESCRIPTION OF A THORACOPLASTY BRACE*

WARRINER WOODRUFF, M.D.
SARANAC LAKE, N. Y.

FOLLOWING thoracoplasty for pulmonary tuberculosis, it is desirable that maximum compression of the side operated upon be obtained before the ribs have regenerated. In securing this, an efficient brace plays an important part. There are many braces and binders in use. The one here described employs well-known principles, but with the exception of the one described by Sauerbruch,† I know of none resembling it, and I believe a short description of it may be of interest, since it gives powerful compression of the side operated upon with minimal restriction of motion of the opposite chest. It is efficient, comfortable, and simple.

The pressure is applied by a pad on the side operated upon extending from the axilla to the lowest rib operated upon. Counter pressure is obtained from a ring around the opposite shoulder girdle and a pad over the crest of the opposite ilium. These two points are fixed, and, regardless of the amount of compression, the movement of the side unoperated upon is not markedly restricted. The amount of compression is regulated by four combined webbing and heavy elastic straps. Two of these straps are in front, the other two are in back. The upper pair extends from the upper part of the compression pad to the ring around the opposite shoulder. The lower pair extends from the bottom of the compression pad to the counter pad over the opposite ilium crest. The iliac pad is prevented from riding up by a perineal strap around the thigh of the same side. If the brace is adjusted to the patient in a sitting posture, it will be found to bind him in the groin when he reclines. He can easily adjust this by loosening the anterior end of the perineal strap and retightening it when he sits up. There are two other straps, one a shoulder strap on the side operated upon to prevent the compression pad from slipping down. This is looped over, and slides along, the posterior strap to the shoulder ring, and it is fastened to the upper border of the compression pad anteriorly. This strap is not used for compression. The remaining strap extends from the lower portion of the shoulder ring to the upper portion of the iliac pad. This is used only when the amount of compression is so great that the ring cuts into the axilla, and it is then tightened enough to pull the ring down out of the axilla.

The compression pad is half thickness soft white felt, covered on the inner side with canton flannel and on the outer with 12 oz. duck rein-

*From the Department of Surgery, University of Michigan.
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†Sauerbruch, F.: Chirurgie Der Brustorgane, ed. 2, 1920, vol. 1, p. 573.

forced by $\frac{5}{8}$ in. strips of 65/1000 in. aluminum. It is approximately $7 \times 8$ in. not including the length of an anterior tongue which fits beneath the clavicle. If made for a woman, great care is used to cut it away over the breast. The shoulder ring consists of a circular strip of 12 oz. duck, 2 in. wide, covered with muslin and padded on the inner side with the felt. The iliac pad is made of 12 oz. duck similarly covered with muslin and padded with the felt. It is about 5 in. in diameter and has five buckles arranged equidistant around the periphery. The two lower ones are for the ends of the perineal strap, the uppermost one is for the strap going to the shoulder ring and the other two are for the

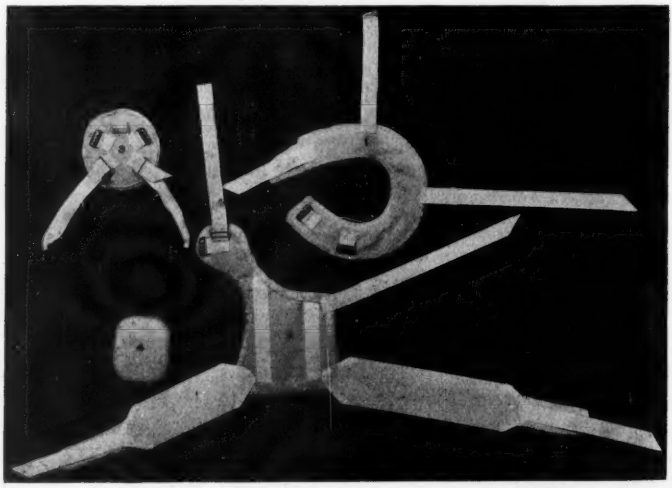

Fig. 1.—Paravertebral thoracoplasty brace before assembling. (1) Shoulder ring; (2) compression pad with straps attached; (3) pad for iliac crest, with perineal strap attached; (4) rubber plug used only after anterolateral stage, pad should be shaped to fit infraclavicular depression due to costal defect.

straps going to the lower border of the compression pad. The perineal strap consists of 1 in. webbing run through $\frac{1}{2}$ in. rubber tubing.

The two straps from the lower edge of the compression pad are of 3 in. elastic sewn to 1 in. webbing which will easily go through the buckles. The two upper straps from the compression pad, and the strap between the shoulder ring and the iliac pad, consist of a double thickness of 1 in. elastic, 3 to 4 in. in length, sewn to 1 in. webbing. Occasionally, for comfort, the shoulder and other narrow straps are padded with half thickness felt. The smaller elastic straps may have to be replaced after a few months, as they are under great tension. The ends of all webbing straps are turned over at an angle of 45° and are sewn to prevent raveling.

If, after a paravertebral thoracoplasty, an anterolateral thoracoplasty

has been necessary to supplement compression of an unusually large pulmonary cavity, the anterior tongue is made larger and a large plug of sponge rubber about 3 to 4 in. in the transverse and longitudinal diam-

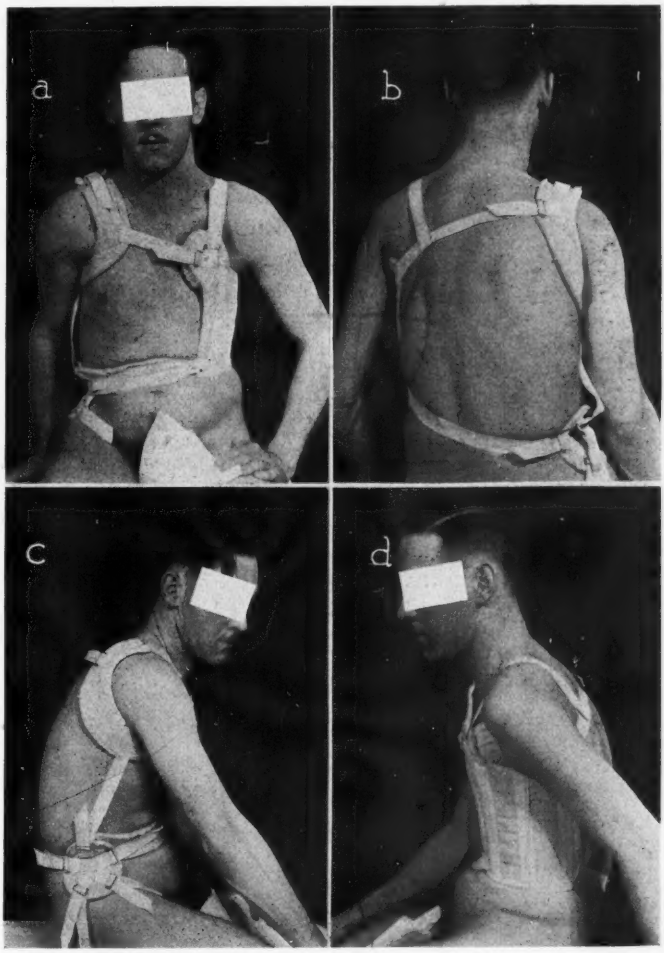


Fig. 2.—(a) Front view of brace used after paravertebral and anterolateral thoracoplasty. Note the left intraclavicular plug used only after anterolateral stage. (b) Posterior view. (c) Lateral view showing the shoulder ring, the position of the counter pad over the iliac crest, and the perineal strap. (d) Lateral view showing infraclavicular plug in place. The shoulder strap is tightened sufficiently to hold the brace in place, but no attempt is made to increase compression through its use.

eters and 2 to 3 in. in depth is placed in the depression beneath the clavicle. The plug is made by cementing together two or three layers of 1 in. sponge rubber, cutting and grinding it to fit the depression and

then covering it with flannel. The first plug consists of two layers, but when it has made a larger depression for itself, another layer is added.

The brace is applied about ten days after the completion of the thoracoplasty and is worn for a period of four to six months, at which time the regenerated bone from the periosteum of the resected ribs has fixed the chest in the collapsed position. The anterolateral brace may have to be worn indefinitely, because the anterior periosteum at times fails to regenerate solid bone.

We wish to acknowledge the help and advice of Dr. John Alexander on whose division this brace was evolved, and of Mr. George E. Curry, who is in charge of the Appliance Shop at the University Hospital.

# Department of Reviews and Abstracts

CONDUCTED BY DUFF S. ALLEN, M.D., ASSOCIATE EDITOR

## Selected Abstracts*

**Felix, A.: Tatigheit des Herzens bei Variation des Druckes über der Herzoberfläche. (Cardiac Action with Variations in Pressure upon the Surface of the Heart.)** Zentralbl. f. Chir. 1931 26: 1664, 1931.

The report is that of a study of the effect of cariations in pressures exerted upon the cardiac surface on the size of the heart and the efficiency of cardiac action. The external pressure factors constantly exerted upon the heart consist in: (a) the tension of the pericardium; (b) the pressure exerted by the chest wall; (c) the elastic tug of the lungs. The problem was to ascertain how variations in these forces can influence both the normal and altered cardiac volumes; if reductions in pressure on the cardiac surface can increase the volume filling of the heart or if increases in the external pressure can cause a diminution in the volume inflow and cardiac volume.

Increased cardiac volume is familiar when cardiac dilatation occurs as an awaited clinic event, but it does not occur acutely and spontaneously to a sufficient degree to be efficient. There is a marked inability of the heart to enlarge rapidly as much as is necessary. Likewise, artificial diminution of volume would have an important rôle when unwanted cardiac dilatation, occurring because of muscular atony and diminution in cardiac power, is to be arrested. A surgically induced relative volume reduction in face of a threatening increase of cardiac volume would be of great service.

A series of experiments was performed to determine the effects of increased or decreased external pressures upon the size of the heart and hence on cardiac action. Decrease in pressure was produced by wide resection of the pericardium, increase in pressure by plication of the pericardium or through exclusion of the influence of the elastic pull of the lungs upon the heart.

Decrease in the external pressure augments cardiac activity when done in the presence of an increase in cardiac volume due to an acute arterial hypertension or an acute mitral insufficiency in the dog, or a chronic arterial hypertension in the rabbit. Acute arterial hypertension can be produced through stenosis of the thoracic aorta. Chronic hypertension in rabbits can be produced through operative elimination of the blood pressure lowering nerves, through section of both depressors and both of Hering's sinus nerves. Acute mitral insufficiency is established through suture of the anterior mitral leaflet to the anterior ventricular wall. These conditions act to increase cardiac volume. Pericardial resection reducing the external pressure in the presence of the increased cardiac volume in these experiments augments cardiac activity. The evidence of the increase in cardiac action is demonstrated by the curves of the carotid pressure, tracings of the intraauricular pressure and computations of the minute volume output.

*These abstracts were made by Dr. Maurice Berck, New York City; Dr. Byron Francis, Chicago; Dr. Herbert Carlson, St. Louis

The influence of an increment in external pressure was determined in rabbits with chronic hypertension. In a large series of previous experiments it has been shown that by extirpation alone of the blood pressure reducing nerves enlargement of the rabbit's heart ensues in about 80 per cent of the cases. After severance of these nerves, increase in the external pressure was secured through abolition of the effects of the elastic recoil of the lungs. The result was that only 15 to 20 per cent of the rabbits' hearts enlarged. An increase in external pressure therefore can arrest cardiac dilatation caused by chronic arterial hypertension in the rabbit.

The experiments with diminution of external pressure show that in the presence of cardiac enlargement increased work can be produced, (pericardial section plus acute hypertension in the dog and rabbit, plus mitral insufficiency and tricuspid insufficiency in dogs.) On the other hand, increase of external pressure can arrest the spontaneous dilatation occurring in hypertensive rabbits.

The critical review of experiments fails to reconcile the conditions produced with disease processes in man because all the experiments were acute and performed in a very short time. The cardiac defects and the acute hypertension were produced in a very short experimental time and not in months and years as occurs in man. No opportunity is therefore allotted for the exertion of any natural compensatory power on the part of the heart in response to the acute experimental changes.

A new method is therefore proposed for the gradual establishment of cardiac defects so that the observed effects of the induced pressure changes on the size of the heart may have an application analogous to ordinary conditions. The cardiac defects produced were mitral stenosis and insufficiency. A suture of autoplastic material (nerve or fascia) is placed between the base of the insertion of the mitral leaflets and the auricular wall. In eight hours a thrombus has formed on the suture, in the course of the next few days it grows larger and by the fifth day has obturated nearly four-fifths of the left auriculoventricular orifice. The result either of stenosis or insufficiency is dependent upon the anatomic conditions of the blockage. The thrombus adheres to the cardiac wall and becomes organized.

This fulfills the postulate that the experimental defect be comparable to the process in man. The natural compensatory powers of the heart can now be exerted, and after their exhaustion, the necessary surgical measures to alter the cardiac volume by pressure increases or reductions can be performed. Only after such experiments have been performed can the results be applied to processes in man.

Alterations in extrinsic cardiac pressure can be effected as easily in man as in animals. Such variations can be induced without resection of the pericardium. The extrinsic pressure can be increased by release of the elastic pull of the lungs by use of a mantling pneumothorax or by use of pressure upon the external precardiac surface. Diminution of external pressure can be secured by resection of the pericardinum or of the bony thoracic cage.

**Stoker, H.: Zur Chirugie der Herzverletzungen und Enstehung der Herztamponade. (The Surgery of Cardiac Injuries and the Origin of Cardiac Tamponade.)** Wien. klin. Wchnschr. **1**: 477, 1931.

The first to successfully operate upon a penetrating wound of the heart was Rehn. Since then three hundred of these cases have been operated upon with a mortality of forty to seventy per cent. Gunshot wounds of the heart are particularly unfavorable both with respect of spontaneous healing (2.7 per cent) and with respect to the injuries and infections of contiguous organs. The prospects for healing and cure of these cases is very low (61 per cent mortality). The author

reports a case of successful operation of gunshot wound of the heart. The clinical picture was that of an overwhelming hemorrhage with shock. The reason for the great internal hemorrhage was a widespread left hemothorax. During the course of the short clinical examination the entire picture changed rapidly. The patient became rapidly worse with symptoms of cardiac tamponade. At operation 350 c.c. of blood was found in the pericardium. The cardiac wound was not bleeding, but in the area behind the heart a well of blood was found arising from hemorrhage from wounded hilus vessels which, through a perforation, was flowing into the pericardium. Postoperatively there developed an empyema which drained spontaneously through the operative incision. The site of the rib resection sank in over a hand's breadth with a systolic retraction and a diastolic pulsion forward—analogous to the condition after a Brauer cardiolysis. Immediately after operation cardiac and respiratory standstill occurred which responded successfully to intracardiac injections of epinephrine. However, for three days ephetonin had to be given to support the circulation.

**Laqueur, B.: Ein Kombinationsklassifikation der Schussverletzungen des Herzens.** Arch. f. Orthop. Chir. **29:** 198, 1931.

This article concerns itself with the assemblage of all the conceivable classifications of heart gunshot injuries with an arrangement for the most adequate classification for analysis, treatment, and disposition. The final classification consists of six groupings:

1. The direction of trajectory of the gunshot and the velocity of a bullet.
2. The amount of hemorrhage.
3. The entrance of air—openings into lungs and pleura.
4. Degree of injury of other chest organs.
5. Degree of infection.
6. Time patient is seen after the injury.

Concerning cardiac wounds, the following classification is made:

1. Tangential.
2. Perforating.
3. Retention of bullet.
4. The retention of the bullet in the pericardium, cardiac walls or within the cardiac cavity. All these groups have subdivisions.

**Levy, R. L., and Moore, R. L.: Paravertebral Injections of Alcohol for the Relief of Cardiac Pain. A Review of Experience to Date and a Report of Nine Cases.** Arch. Int. Med. **48:** 146, July, 1931.

This study is a critical appraisal of the results to date of the paravertebral injections of alcohol (57 cases reported in the literature, and the record of personal observations in 9 cases).

Sensory fibers in the heart form a plexus surrounding the coronary vessels and aorta and in this plexus minute sympathetic ganglia are found. Afferent impulses are carried to the spinal cord along the cardiac branches of the middle and inferior cervical sympathetic ganglia (middle and inferior nerves) and pass through the sympathetic trunk and into the upper five (possible six) thoracic nerves through the corresponding white rami communicantes. Afferent impulses from the heart are also carried to the brain along the vagus and may cause pain referred to the head through communicating branches with the III, IV and V cranial nerves. No afferent fibers from the heart to the superior cervical sympathetic ganglion have been demonstrated, and for the present the superior cardiac nerve must be regarded

as entirely motor. In certain cases relief from painful paroxysms has been obtained following the removal of only the superior cervical sympathetic ganglion. To explain this the hypothesis has been advanced that vasoconstrictor fibers reach the cardiac plexus from this ganglion by way of the superior cardiac nerve—causing painful seizures through coronary spasm. These cases at necropsy may show no morbid changes within the heart.

In view of the number of afferent pathways from the heart to the cord and brain and with the possibility of individual variation from a standard pattern being taken into account, the difficulty of interrupting all of these pathways by cervical sympathectomy becomes apparent. On theoretical grounds, paravertebral alcohol injections should afford a better chance for success with less trauma, as they interrupt the passage of painful impulses at their point of entrance to the spinal cord. However, impulses can still be transmitted to the cranial nerves through the vagus and vaso-constriction of the coronaries may still be possible via the superior cardiac nerve. There are probably other as yet uncharted pathways.

The technic described is that of Labat as modified by Swetlow and White. No attempt was made to determine into which roots injections should be made by mapping areas of disturbed sensibility of the skin. It was frequently impossible to delimit certain levels, and it was believed that by making injections into the first five dorsal rami, the main known sensory pathways to the cord were interrupted.

An estimate of the degree of the relief obtained was made by averaging the results as recorded in 49 cases including the 9 cases observed by the authors. Complete or almost complete relief was obtained in 51 per cent of the cases, improvement was noted in 34 per cent and failure in 15 per cent. Some patients were benefited only slightly and temporarily; in others the result was strikingly successful. In one personal case there was a complete relief from pain for 16 months.

After injection, a majority of patients suffered from hyperesthesia of the chest wall and painful intercostal neuritis in the segmental distribution of the nerves into which injections were made. At times the discomfort lasted as long as six weeks and was apparently unavoidable. Many patients had fever following injection, lasting from a few days to a week. In two of the personal cases effusion into the left pleural cavity followed the injections.

Horner's syndrome was observed in 7 of 15 cases in which notes on this point are available. Its occurrence did not afford a therapeutic index of the ultimate result. It tended to disappear in the course of several weeks or months.

Paravertebral alcohol injection offers a reasonably good hope of some relief to patients with paroxysmal cardiac pain. Final judgment as to its value and limitations must be reserved until longer observations have been made. It should be tried only after carefully planned medical treatment has failed to alleviate intense suffering. It is relatively safe and has been attended by no operative fatality. It has a sounder physiologic basis and is less dangerous than cervical sympathectomy. However, being entirely symptomatic, it cannot alter the basic pathologic condition. Patients to whom pain has served as a danger signal of overexertion should be warned, if relieved of attacks, against exceeding the functional capacity of the heart.

**Carajannapoulos, G., and Lazarides, P.: Kystes Hydatiques du Poumon. (Hydatid Cysts of the Lungs.)** J. d. Chir. 37: 539, April, 1931.

In Greece the incidence of pulmonary hydatid is relatively large, one in every 175 operations for hydatid cyst. Twelve cases operated upon in the last three years are analyzed. Weinberg's reaction is unreliable in the majority of cases. More confidence is placed on Casoni's skin reaction and upon eosinophilia.

Casoni's reaction particularly in non-suppurating cysts has proved of great value. Operative indications for hydatid cyst are not present in the small, centrally located, hilar cysts which frequently break into the bronchial tree and thus cure themselves by spontaneous evacuation. Operative indication is present, however, in (a) those cases of centrally located cyst which present severe hemoptysis, (b) those which after rupture are the cause of a manifest aggravation of the general condition, (c) in cysts larger than an orange, (d) peripheral cysts, (e) intrapleural cysts.

Local anesthesia is used. Rib resection of 6-8 cm. at the site of election is performed. In the majority of instances pleural adhesions permit the performance of a one stage evacuation of the cyst. Formaldehyde injections are not done. The cyst is usually found at a depth of 1-2 cm. and is entered with a bistoury, the incision in the cyst is not more than 4 cm. long and is always sufficient to extract the lining membrane and its contents. In eight cases where such adhesions were present the operation was simple and well-borne, the only complication being a severe coughing spell sometimes attended by hemoptysis when the cyst lining is extracted. If pleural adhesions are absent the danger of spilling the hydatid fluid or pus into the free pleural cavity is considerable. Anaphylactic accidents are not so common as we have been led to believe but purulent and echinococcus pleuritis are dangerous complications. The usual technic employed consists in (a) progressive and gentle induction of the surgical pneumothorax, (b) direct exploration of the lung with the attempt to fix the most superficial portion of the cyst at the center of the operative field, (c) meticulous walling off of the free pleural cavity, (d) mobilization of the cyst to the outside followed by evacuating puncture, (e) opening of the cyst and ablation of the lining membrane and its contents, (f) finally fixation of the sutured walls of the cyst to the parietes. If suppuration is not present the above one stage technic in the absence of adhesions is always employed. If radical operation is contraindicated, phrenicotomy for hydatid cyst of the lower lobes may be performed. In one case an apparently good result was obtained. Factors preventing closure of the cavities present after ablation of the lining membrane are (a) latent or secondary infection due to the bronchial communication frequently present, (b) the large dimensions of the cavity, (c) great thickness of the wall, (d) inelasticity of the surrounding pulmonary parenchyma, (e) finally the location of the cavity, those at the apex resisting obliteration longest. If the cyst contents at the time of operation are clear, provisionary drainage is instituted by a small calibered tube around which the lips of the incision are hermetically sutured. The tube is tied off and if in 5 to 6 days convalescence has been symptomless and apyretic it is withdrawn and primary union takes place. If fever ensues because of infection within the cavity, the tube is opened and drainage is begun. Some cavities are, because of infection, packed open widely at the time of operation. Postoperatively empyema is occasionally noted which is treated by rib resection. Bronchial fistula are usually obliterated by the lung expansion. The detailed report of 12 operated cases is appended.

**Plieninger, T. Uber Lungenaktinomykose. (Primary Pulmonary Actinomycosis.)** Ztschr. f. Tuberk. **69:** 152, 1930.

A brief report is made of the general bacteriologic characteristics of actinomycosis. Primary pulmonary actinomycosis is quite rare amounting to only 10 to 15 per cent of all the cases, while cervical and maxillary lesions constitute 70 per cent of the cases and intestinal lesions 20 per cent.

Secondary pulmonary actinomycosis occurs as a metastatic lesion by way of the blood or lymph stream, when the primary lesion is situated some distance from the lung. When invasion is by contiguity from a lesion in the vicinity it is indistinguishable from primary pulmonary actinomycosis.

The organism can directly invade the lung through inhalation of vegetable dust as in cases seen in workers with cereals. But this pathogenesis is rare. This inoculating lesion disappears without leaving a trace and the lung is reinoculated by way of the blood stream or lymphatics. Some authors claim that the unrecognized primary lesion is in the tonsil and is the origin of the generalized infection.

The mycosis forms a chronic suppuration which is unrecognizable without the presence of the characteristic yellow granules. The pulmonary lesion consists of a round-celled infiltration with central necrosis and a perifocal inflammatory reaction. It causes widespread excavations associated with massive sclerosis and induration.

The clinical picture has no definite diagnostic characteristics until the pulmonary process erodes the thoracic wall and appears under the skin. The subcutaneous induration has a ligneous consistency, often a violaceous bronzed hue, ulcerates and soon fistulates. The fistulas remain open, or surrounded by exuberant granulations and present a sarcomatous appearance. The pus that exudes has a stale odor and usually contains the characteristic yellow granules.

One may distinguish two different localizations of the disease: a hilar form which goes on to peribronchial infiltration penetrating into pulmonary parenchyma and causing a widespread intense pulmonary sclerosis, and (2) a primary parenchymal localization when the lesion presents itself as a localized pulmonary infiltration, as a lung abscess, an infarct or a localized bronchopneumonia. The lower lobes are most often attacked and pleural complications are inevitable.

In the course of the clinical evolution, three stages may be distinguished:

1. Stage of bronchopneumonia. Cough with mucopurulent, occasionally bloody, expectoration. Signs of persistent bronchitis. Fever and loss of weight. No radiological signs. This is the bronchitic form but if parenchymal changes appear radiological signs become manifest.

2. Stages of pleuritic involvement. The process reaches the pleura. Severe pleuritic pain often present. Effusion may occur. Occasionally retraction of the thoracic wall. Progressive insidious course but often with high fever and chills. Soon there occurs an invasion of the parietes.

3. Stages of Fistulas. Characterized by invasion and ulceration of the parietes with fistulae formation and the evacuation of actinomycotic pus. Progressive downhill course with severe cachexia. Metastatic lesions appear in the other viscera. Amyloidosis ensues with marasmus. Cardiovascular insufficiency precedes death which occurs i a few months, but rarely not for several years after the onset of the disease.

Diagnosis is usually not made until the third stage is reached. All the cases diagnosed in the past twenty-five years have succumbed.

In cases of diagnosis made in the earlier stages some hope may be held out with vigorous iodine therapy, intensive radiotherapy and, perhaps in well localized cases, surgical treatment.

Early cases might be diagnosed by the presence of actinomyces in the sputum. The various humoral reactions are of doubtful value (complement fixation, and intradermal reactions). The chief differential diagnosis is from tuberculosis.

**Denk, W., and Domanig, E.: Chirurgie der Lungentuberkulose. (Surgery of Pulmonary Tuberculosis.)** Beitr. z. Klin. der Tuberk. **77**: 3, May, 1931.

The article is based upon twelve years' experience with 175 thoracoplasties, 142 phrenicexereses and 30 extrapleural paraffin plombe pneumolyses at the Graz surgical clinic. Great care must be taken in the evaluation of statistics in the surgery of tuberculosis because of the long remissions and the chronicity of the disease. The indications for surgery can only be relative and cannot be set down in an absolute manner. The problem of the length of time taken for conservative treatment before surgical therapy is employed is of the greatest importance. Exact indications cannot be set down because of the great variety of clinical courses. In general, if the phthisis, in spite of sanatorium care, is progressing without a natural spontaneous tendency to cure one should substitute surgical for expectant treatment. Besides the well-known contraindication for surgery of an acute exudative lesion, the presence of a badly damaged heart with a severely compromised circulatory status has been found to exert most influence in prohibiting surgery and in giving a bad prognosis. Much of the morbidity and mortality of thoracoplasty is due to the effects of the operation upon a damaged circulation. Minor degrees of circulatory impairment in cases requiring operation are treated with continuous intravenous infusions during the operation. Careful attention must be paid to the pulse and blood pressure. The operation is performed in as many stages as seems necessary. The earlier and more complete the surgical procedures are, in cases that are progressing and showing no natural tendency to cure themselves under expectant treatment, the better will be the final results. Social consideration may encourage surgical therapy to return the patient to his livelihood sooner than could be expected under expectant treatment. Unsatisfactory pneumothorax with basilar gas and adhesions blocking off the pleural space over the upper lobe lesions is a prime indication for surgical therapy. In cases of bilateral lesions, operation is performed only after the better side has been shown to be inactive and not spreading. Upper lobe lesions of the opposite side have been seen to disappear after successful surgical compression of the more affected side. However, the status of the "better" lung is of paramount importance in the consideration of surgical therapy and should not be lightly dismissed. Tuberculosis of other organs does not always contraindicate surgical therapy. Tuberculosis of one kidney, of the ileocecal region, of the larynx or isolated osseous lesions are not contraindications. In this series five patients with laryngeal tuberculosis had total thoracoplasties performed of which two are cured for the past four years and two are greatly improved. One patient with lingual tuberculosis has been well for eight years following total thoracoplasty. A patient with tuberculosis of the prostate and epididymis withstood the operation well but died four months after total thoracoplasty with disseminated tuberculosis. The absolute contraindications are:

1. A badly deteriorated general condition, especially a badly embarrassed heart with a poor circulatory status.
2. Rapidly progressive exudative lesions, especially caseating pneumonia.
3. Multiple tuberculous lesions in various organs.
4. Widespread intestinal tuberculosis.
5. Bilateral renal tuberculosis.
6. Severe amyloidosis.
7. Progressive disease or emphysema of the opposite side.
8. Patients over sixty years old.

Thoracoplasty was performed 175 times with an immediate operative mortality of 16.6 per cent and a late mortality of 21.2 per cent. The indications for a total

thoracoplasty are present when there is a predominatingly unilateral lesion widespread on that side. As contraindications in this type there are:

1. Very severe deterioration of general condition particularly very severe cardiac and circulatory damage.
2. A rapidly exudative progressive downhill course never responding to rest therapy.
3. Severe degree of tuberculous and nontuberculous disease of other organs.
4. Patients over sixty.

Paravertebral thoracoplasty affords a degree of pulmonary collapse only commensurate to the number and length of the ribs resected. The thoracoplastic resection must always be selective, adapted to the position and size of the cavities underlying the operative site.

Total thoracoplasty can usually be performed in two stages but in cases with circulatory impairment three or more stages may be employed.

In addition to local anesthesia a light general anesthesia may be employed without danger.

Pulmonary and circulatory complications are the greatest causes for postoperative mortalities. Patients with right-sided lesions, with poor general condition and labile circulation, with exudative phthisis of a severe progressive character with widespread cavity formation, are especially prone to postoperative complications.

A lasting good result depends most of all upon a sufficient degree of compression of the lesion, upon the status of the "better" side, and upon the nature of the severity and the tendency to spread of the lesion itself. Appropriate postoperative sanatorium care of sufficient duration is of the greatest importance.

The follow-up of the subsequent course of patients treated with thoracoplasty is essential. In many cases in which the effect of thoracoplasty has not been quite satisfactory, additional supplementary plastic operations have been successful.

Secondarily infected tuberculous pyopneumothorax must be operated upon as soon as practicable. Open thoracotomy with rib resection as a preliminary operation to thoracoplasty has been quite satisfactory.

Partial thoracoplasty (resection of the first eight ribs) in lesions confined to the upper lobe is performed in conjunction with phrenicexeresis.

Extrapleural pneumolysis with paraffin plombe has not been satisfactory. It has been performed in thirty cases of which seven died postoperatively and seven have been improved. It is considered unsatisfactory on these grounds:

1. Operation was performed only in cases where the lesion was restricted to the upper lobe and in whom the prognosis with partial thoracoplasty plus phrenicexeresis is usually good; yet the mortality was excessive (23 per cent) and only 23 per cent were improved.
2. Efficient compression of cavities is not always secured, particularly in cavities situated close to the hilus.
3. In two cases severe infection of the plombage bed ensued with extrusion of the plombe.
4. In two cases irruption of the plombe into the cavity took place with eventual external fistula formation.
5. The incidence of spill-over infections of the lower lobes and of circulatory failure postoperatively was unduly great considering the mild restricted type of case operated upon.

Phrenicexeresis (142 cases) should be performed only by experienced operators to avoid injury to other nerves. It is suitable to improve the collapse brought about by other measures. Used independently it only rarely can produce a lasting curative

effect. It can most suitably be employed in combination with partial thoracoplasty for lesions of the upper lobe.

In severe widespread phthisis phrenicexeresis is often the only form of collapsotherapy available to favorably influence the lesion.

With recently acquired mild lesions satisfactory results can be obtained with a combination of phrenicexeresis and pneumothorax. Also without simultaneous pneumothorax the results of phrenicexeresis in the mild recently acquired lesions are often surprisingly satisfactory.

**Bernard, L. & Poix, G.: La Phrenicectomie dans le Traitement de la Tuberculose. (Phrenicectomy in the Treatment of Tuberculosis.)** Presse med. 39: 201, February, 1931.

The authors present an analysis of sixty cases of phrenicectomy performed for pulmonary tuberculosis. Thirty-five of the cases were hospitalized, twenty-five others were operated upon without hospitalization. In all the cases operation consisted of phrenicexeresis done under local anesthesia. No operative or postoperative complications occurred.

Six cases operated upon presented a slight dyspnea which tended to disappear within a few weeks. Three who had hemoptysis preoperatively had hemoptysis postoperatively but never of any great severity. In two cases performed on the left side in instances of pneumothorax, there were mild postoperative cardiac discomforts which tended to disappear within a few months. On six occasions there were postoperative exacerbations of a contralateral process. In all the cases there were bilateral lesions strongly predominant on one side and appearing inactive on the other. In three cases exacerbation of the lesion went on to death. It is therefore certain that phrenicectomy is not always devoid of danger and when employed as a test operation before thoracoplasty, its results may not be benign.

In fifty-six cases the length of the nerve avulsed varied between 5 and 36 cm. and in all the cases except one diaphragmatic paralysis was complete. In four cases 4 cm. or less than 4 cm. was avulsed and in two of these cases paralysis was incomplete, in the third there was an immobilization of the diaphragm without ascension and in the fourth ascension only attained three cm. It is advised therefore to extract at least five cm. of the nerve. Paralysis includes both immobilization and ascension. Immobilization is usually immediate and complete. Ascension varies with a degree of paralysis, when paralysis is complete it usually immediately follows operation. It varies between two to ten cm. and sometimes later attains its highest level. The rise is conditioned in part by the abdominal pressure and in part by the pulmonary contraction—having factors in the loss of physiologic elasticity of the healthy diaphragmatic muscle and in the secondary pathologic retraction of the diseased lung.

Phrenicectomy acts comparably to a pneumothorax. The relaxation of the lung produces a diminution in respiratory activity, in circulation of blood and lymph and favors the production of cicatricial contraction. But because of the small degree of collapse it causes, it can never create a sclerosis of the lung. Cough diminishes after operation, expectoration usually increases for ten to twenty days, then lessens and may even disappear. Each time that a case seemed to present a lasting improvement a diminution and even a cessation of expectoration was seen. Each time that the status of the case remained stationary, the diminution in expectoration was transient and later resumed its former proportions or was increased.

In ten cases out of forty-five the bacilli disappeared from the sputum; these were all circumscribed, localized lesions with or without cavities. The bacilli did not disappear in those with widespread diffuse lesions. The fever, if it was at all

elevated, was not immediately influenced, but if the preoperative temperature was never above 38° it often returned to normal.

Ordinarily improvement was rapid and manifest in the first month. If the patient did not receive much benefit in the first six months, the chances of a notable improvement setting in later were minimal.

In forty-five cases, phrenicectomy was performed independent of any other method. These cases were in chiefly unilateral lesions in which pneumothorax was unsuccessful because of pleural adhesions. In only four cases was phrenicectomy employed at the onset in preference to pneumothorax. In one group the authors arrange a series in which a solitary cavity was situated in some part of the lung. The majority of these cases had undergone sanatorium treatment without a diminution in the size of the cavities or disappearance of tubercle bacilli in the sputum. In eight out of nine cases the result was favorable. In five cases a clinical cure was obtained.

In a second group of cases (21) the lesions were more diffuse and further advanced, predominatingly ulcero-fibrous with a manifest tendency to retractile sclerosis, in which pneumothorax was impossible because of pleural symphysis. In six cases marked improvement occurred in the first week and went on to clinical cure. These six cases were well advanced but circumscribed lesions. In eight others, cases with diffuse lesions there was only a functional improvement. In the remaining seven, signs of marked retraction were present but the lesions progressed, the fever remained high and the best result in this group was a transitory amelioration.

The last group consisted of fourteen cases of the ulcero-caseous type without any tendency to fibrous retraction. In five cases who had shown no signs of progressive activity—the results were moderately good; in the remaining nine cases with high fever the only result was that in certain cases a moderate defervescence occurred.

In consideration of the results the authors state that success with phrenicectomy is conditioned by three primary factors:

(1) The tendency to a fibrous retractile process (which is indispensable to success).

(2) The extent of the lesion. The chances for success are much greater with circumscribed lesions. The size of a lesion is much more important than its site in estimating the probable success of phrenicectomy.

(3) The pathological nature of the lesion. Those lesions that are progressing rapidly do not respond well.

The association of phrenicectomy with pneumothorax does not diminish the frequency of refills nor has it lessened the incidence of pleural reactions to pneumothorax. The authors do not therefore recommend the use of phrenicectomy when a pneumothorax can be successfully induced. If, on the contrary, pneumothorax is unsuccessful because of adhesions, the indications vary with the type of case, i.e., in cases of adhesions to the apex and to the upper parieties, phrenicectomy has nothing to offer. If, on the contrary, pneumothorax is prevented because of diaphragmatic adhesions, phrenicectomy is indicated and very useful.

The collapse produced by phrenicectomy is considerably less than that evoked by pneumothorax and its chances less therefore of provoking an exacerbation or evolution of a lesion in the contralateral lung.

The majority of authors are in favor of phrenicectomy prior to thoracoplasty. The author believes a month should elapse between the two procedures. One can then utilize phrenicectomy as a complementary operation instead of the usual synchronous association of the operations.

Phrenicectomy should not be performed as an independent measure unless a pneumothorax is impossible or inefficacious. It should be reserved as an independent procedure only for those cases which seem to have the best spontaneous prognosis.

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## In Memoriam

### CHARLES DANIEL LOCKWOOD

Charles Daniel Lockwood died suddenly at his home in Pasadena, California, on June 11, 1932, of erysipelas.

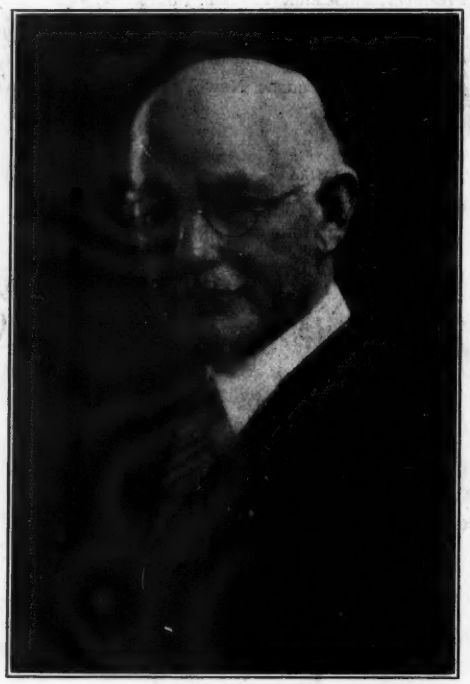
Dr. Lockwood was born in Effingham, Illinois, January 22, 1868. He received his A.B. degree from Northwestern University in 1893 and his M.D. from the same university in 1896. The following year he did postgraduate work in Vienna. His internships were served in the Chicago Lying-In and Cook County Hospitals.

In 1898 he married Clara M. Sanford of Platteville, Wisconsin, who survives him.

He served as assistant instructor in surgery at the Northwestern University Medical School 1897-99. Alternate member of California State Board Medical Examiners 1901-02. Professor of Surgery, College of Dentistry University of Southern California 1902.

He rapidly rose to surgical prominence in the Southwest and then to national recognition. He organized the Red Cross Ambulance Company No. 1. He entered the Great War as a Major M.C., U. S. A., and retired from service in 1919 as Lieutenant-Colonel. He was a member of the Western Surgical Association, one of the founders and first president of the Pacific Coast Surgical Association, and a charter member of the Editorial Board of the Western Journal of Surgery, Obstetrics and Gynecology.

His interest in the advance of general surgery was great. He was always among the first to recognize improvements in surgical procedures and conceptions. Following an extensive experience in chest injuries in France he returned to civil practice convinced that thoracic surgery would advance with amazing rapidity and success. He was at once appointed chest surgeon for the Olive View Sanatorium where he continued until his death. Since that time his chief surgical interest had been in this branch of his work. He attended regularly the meetings of the



CHARLES D. LOCKWOOD
1868-1932

American Society for Thoracic Surgery and contributed to its development.

In the last years of his life he labored as steadily as he was physically able, harder than he should have.

Dr. Lockwood was dearly beloved in his community and respected throughout his country. He strived always to fulfill in the daily practice of his profession the high duties which he felt were expected of him. He devoted his life conscientiously, steadily, constantly to the advancement of surgery. He was always pleasant, considerate and earnest. His word of cheer, his kindly smile, his contagious enthusiasm will be sorely missed.

FRANK S. DOLLEY